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Environmental Assessment for the Satellite Power System-Concept Development and Evaluation Program-Microwave Health and Ecological Effects

November 1980

Prepared for:
U.S. Department of Energy
Office of Energy Research
Solar Power Satellite Project Division
Under Inter-Agency Agreement AI01-79ER10035

DOE/NASA
Satellite Power System
Concept Development
and
Evaluation Program

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Environmental Assessment for the Satellite Power System-Concept Development and Evaluation Program-Microwave Health and Ecological Effects

November 1980

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NASA/DOE
Satellite Power System
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and
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1 INTRODUCTION

The U.S. Department of Energy (DOE) is considering several options for generating electrical power to meet future energy needs. One of these options, the satellite power system (SPS), would collect solar energy with a system of geosynchronous satellites in space, each of which would convert the solar energy to microwave energy and transmit the microwave energy from a directive antenna to a large, earth-based receiving/rectifying antenna (rectenna) for that satellite. A reference system has been described that defines many of the component subsystems of such a concept (US DOE and NASA, 1978). The potential environmental impacts associated with constructing and operating the SPS are being assessed as a part of the DOE's SPS Concept Development and Evaluation Program.

This report is concerned with the potential health and ecological effects of the microwave beam from the microwave power transmission system (MPTS) of the SPS. The report is written in the form of a detailed critical review of selected scientific articles from the published literature on the biological effects of nonionizing electromagnetic radiation, followed by an assessment of the possible effects of the SPS, based on exposure values for the reference system (U.S. DOE and NASA, 1978).

In various parts of the report, radiofrequency radiation is referred to as "RFR," "microwave radiation," "microwaves," or "radiation." Although the terms are used somewhat interchangeably, "microwaves" or "microwave radiation" appears most commonly in Chapter 6, where most of the studies were performed in the microwave region, and "radiofrequency radiation" or "RFR" appears most commonly in other chapters, particularly Chapter 5, where studies include a broader range of the electromagnetic spectrum.

1.1 THE PROBLEM

The microwave energy generated at each satellite will be transmitted by the MPTS to earth in the form of a relatively narrow beam of continuous-wave (CW) RFR having a frequency of 2.45 GHz. The power-density profile of the beam at ground level (and within the troposphere) is illustrated in Figure 1-1, and the power densities at various locations along the path of the beam are as follows (USDOE and NASA, 1978):

- center of transmitting antenna, 2.2 W/cm^2
- edge of transmitting antenna, 240 mW/cm^2
- peak near-field pattern density (on-axis, 1600 km from transmitting antenna), 3.2 W/cm^2
- center of rectenna, 23 mW/cm^2
- edge of rectenna, 1 mW/cm^2
- rectenna site exclusion boundary, 0.1 mW/cm^2
- first sidelobe level (approximately 9 km from center of rectenna), 0.08 mW/cm^2
- grating lobe levels (at spacings of 440 km), $\leq$ 0.01 mW/cm^2.

Therefore, the basic RFR exposure situations are:

(a) People may be nonoccupationally exposed for long periods to power densities generally much less than 0.1 mW/cm^2 because they reside or work in population centers near a rectenna site but outside the exclusion boundary of the rectenna.

(b) Persons entering the rectenna site for occupational reasons could be exposed to power densities of up to 23 mW/cm^2 (and perhaps higher if reflections are considered) unless suitable safety precautions are invoked.

(c) Space workers would be occupationally exposed to considerably higher power densities (e.g., of the order of W/cm^2) if their space suits do not include adequate shielding from RFR.

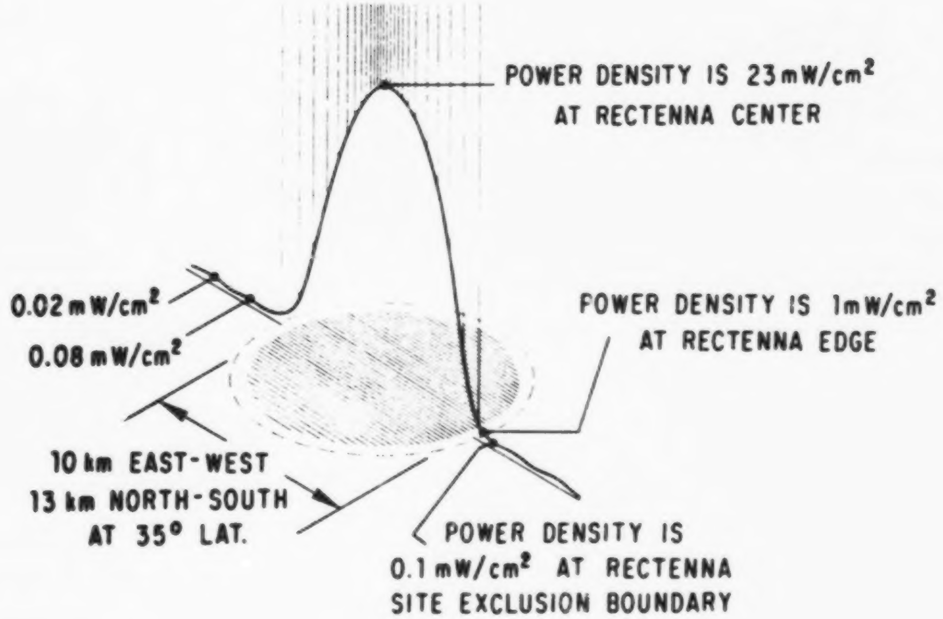
POWER DENSITY IS 23 mW/cm²
AT RECTENNA CENTER
0.02 mW/cm²
0.08 mW/cm²
POWER DENSITY IS 1 mW/cm²
AT RECTENNA EDGE
10 km EAST-WEST
13 km NORTH-SOUTH
AT 35° LAT.
POWER DENSITY IS
0.1 mW/cm² AT RECTENNA
SITE EXCLUSION BOUNDARY

FIGURE 1-1 SPS MICROWAVE POWER DENSITY CHARACTERISTICS AT RECTENNA SITES (from USDOE, 1980)

(d) Airborne biota (birds, flying nonhuman mammals, insects) within the main MPTS beam will be exposed to power densities of up to 23 mW/cm^2 for durations that depend on whether they are transient or indigenous to the rectenna site.

(e) Humans flying through the main beam in airplanes will be exposed to power densities generally much lower than 23 mW/cm^2 because of RFR shielding by the metal skin of the fusilage. It is possible that RFR will enter the fusilage through windows. However, the maximum incident power inside the cabin will not exceed the maximum value just outside the aircraft because the cabin and its contents (including the humans) will constitute an extremely lossy (low Q) multimode resonant cavity.

(f) Plants growing in the rectenna area will be exposed to power densities ranging from 0.01 to 1.0 mW/cm^2. Plants growing outside the rectenna area will be exposed to power densities of up to 0.1 mW/cm^2, but generally much less.

(g) Non-airborne animal species will be exposed to power densities of 0.1 mW/cm^2 or less outside of the rectenna area and between 0.01 and 1.0 mW/cm^2 within the area, provided that they remain below the rectenna. If they climb on top of the rectenna, they will be exposed to 23 mW/cm^2.

1.2 DATA BASE AND LITERATURE SELECTION

Many sources were used in acquiring a working data base for this assessment, including reference bibliographies provided in previous reviews of the literature; a comprehensive bibliography prepared by U.S. Government personnel; published proceedings of recent seminars and meetings on the biologic effects of RFR; the computerized data base on Biological Effects of Electromagnetic Radiation (BEER file) of the Mead Technology Corporation, Dayton, Ohio; and compilations of articles published by the Franklin Institute. Consideration was also given to recent symposia on the biological effects of RFR.

Several criteria were used in selecting articles for inclusion in this review. Preference was given to complete papers published in scientific journals or proceedings of scientific symposia. Where details of the procedures and findings were sufficiently clear and complete, abstracts of presentations at recent scientific symposia were also used. Considerations included the date of publication (more recent articles were preferred because of improvements in the technology of exposure and dose measurement), the frequencies of the RFR (especially frequencies close to those of SPS, but also others in the general range from 10 MHz to 18 GHz as appropriate), and the significance of the findings to human health (e.g., studies of human populations to ascertain whether the occurrence of specific effects is statistically higher in population samples exposed to RFR than in similar population samples not exposed, and experiments involving long-term exposure of animals). Other criteria included the relevance of an article to others on the same topic and possible relevance to concerns expressed by citizens' groups. The number of articles selected was necessarily limited because of the large number of references on the biologic effects of RFR. However, the articles selected are representative of the entire body of literature on this subject.

1.3 EASTERN EUROPEAN BIOEFFECTS LITERATURE

Probably the most controversial aspects of research on the biological effects of RFR are the large discrepancies between results, at low levels of RFR, reported in the Eastern European literature and those obtained in Western countries such as the United States, and the basic differences in philosophy between the two groups of countries in prescribing safety standards or guidelines for the protection of humans against possible hazards from exposure to RFR.

From the end of World War II to about the late 1960s, few of the scientific reports on bioeffects research in the USSR (or other Eastern European countries) were amenable to critical review because they lacked essential information. In the early 1970s, starting essentially with an international conference on the bioeffects of RFR in Warsaw in 1973 under the joint sponsorship of the World Health Organization (WHO), the U.S. Department of Health, Education, and Welfare (HEW), and the Scientific Council to the Minister of Health and Social Welfare of Poland, international interchanges of information increased materially, and translations of Eastern European articles became easier to obtain. Because most of the Eastern European documents prior to 1973 (and many since then) are merely abstracts that contain no details of the experimental method, number of subjects, or analytical approach used in the study, evaluation of them proved difficult. More recent Eastern European studies contain more detail and number of them are cited in the bibliography.

2 PRESENT CLIMATE AND CONTEXT

2.1 PROLIFERATION OF RFR SOURCES

The SPS concept has been introduced in a general climate of widespread proliferation and usage of RFR-emitting devices and systems. Demand for the services and facilities of these devices is reflected by the following figures for the United States (McRee, 1978). During 1976, sales of communications and electronics products are estimated to have totalled $38 billion. The FCC, as of 1977, had authorized transmissions by over 9 million transmitters. Between 1971 and 1978 there was an increase of 87% in the number of FM stations operating in the 88-108 MHz band. The National Institute of Occupational Safety and Health (NIOSH) has estimated that over 35 million industrial RF sources for heating and drying are in use in the plastics, paper, and other industries. Approximately 5 million microwave ovens have been installed in homes in the United States (McConnell, 1978). As of 1979, about 30 million citizen band (CB) radios are licensed, with those operating in the 27-MHz band capable of emitting up to 4 watts. Domestic and business satellite communications systems are burgeoning. Air and maritime navigation makes widespread use of fixed and mobile radar systems.

Thus, there is a widespread climate of acceptance of the benefits of RFR devices for communications, radar, and industrial processes. On the other hand, there are concerns whether the proliferation of usage of RFR devices including SPS may be associated with some as-yet-undefined hazardous biological effects. The purpose of the present document is to address such concerns as they pertain to the SPS.

2.2 MEASUREMENTS OF ENVIRONMENTAL LEVELS OF RFR IN SELECTED U.S. CITIES

The Environmental Protection Agency (EPA) is measuring the environmental field intensities at selected locations within various U.S. cities. Tell and Mantiply (1980) and Janes (1979) discuss the results for the 15 cities (total of 486 sites) studied so far. The sites in each city were selected so as to permit estimations of cumulative fractions of the total population being exposed at or below various average power densities, based on the population figures for the 1970 census enumeration districts.

Field intensity measurements were made at 6.4 m (20 ft) above ground at each site in the following frequency ranges (Janes et al., 1977): 0.5 to 1.6 MHz (the standard AM-radio broadcast band), 54 to 88 MHz and 174 to 216 MHz (the VHF-TV bands), 88 to 108 MHz (the standard FM-radio broadcast band), about 150 and 450 MHz (land-mobile bands), and 470 to 890 MHz (the UHF-TV bands). The signals in each band were received with separate antennas designed specifically for each band. However, the measurements in the standard AM-radio broadcast band were not included in the analyses because this band is below the 10-MHz lower frequency limits of the present U.S. radiation protection guideline (ANSI, 1974).

The measured field strengths at each site were integrated over the frequency bands from 54 to 890 MHz included in the analyses and converted into equivalent average power densities. The site values in each city were then used with the population figures in the various census enumeration districts in a statistical model designed to estimate the population-weighted median exposure value for that city and to calculate other statistics of interest.

The population-weighted median value for a city is defined as the average power density at or below which half the population of the city is being exposed. The results are based on the assumption of continuous exposure of people where they reside; they do not take into account population changes since the 1970 census, population mobility, exposure at heights greater than 6.4 m, attenuation of signals by buildings, or periods of time when any of the contributing RFR sources are not transmitting. These median values range from 0.002 μW/cm^2 (for Chicago and San Fracisco) to 0.020 μW/cm^2 (for Portland, Oregon). The population-weighted median for all 15 cities is 0.0048 μW/cm^2. Also, the percentage of the population of each city exposed to less than 1 μW/cm^2 ranges from 97.2% (for Washington, D.C.) to 99.99% (for Houston, Texas), with a mean value for all 15 cities of 99.4%. The major contributions to these exposure values are from the FM and TV broadcast stations.

EPA also made measurements at sites close to single or multiple RFR sources, e.g., at the bases of transmitter towers and at the upper stories (including the roof) of tall buildings or hospital complexes close to transmitter towers. At the base of an FM tower on Mt. Wilson, for example, the fields ranged from 1 to 7 mW/cm^2 (Tell and O'Brien, 1977, cited in Janes, 1979), but such values are believed to be uncommon. Most measurements in tall buildings close to FM and TV transmitters yielded values well below 100 μW/cm^2, but a few values were close to or slightly exceeded 200 μW/cm^2 (e.g., 230 μW/cm^2 on the roof of the Sears Building, Chicago).

Janes (1979) also discussed the field intensities close to ground-based transmitters of satellite communications systems, radars used for air-route surveillance and other activities, microwave radio relay transmitters, microwave ovens, and personel radios (CBs). He also mentioned other sources such as those used for medical applications (diathermy, electrosurgery) and for industrial applications (heating, drying, and sealing).

Because rectenna sites are likely to be distant from major population centers such as those analyzed in these studies, the increases in population exposure values in these and other densely populated regions due to operation of SPS are likely to be very small. However, when the locations of the rectennas are specified, it would be pertinent to determine the ambient exposure values over the entire RFR frequency spectrum due to the SPS and other RFR sources in the municipalities closest to each site.

2.3 EXPOSURE STANDARDS

The term "exposure standards" is generally applied to specifications or guidelines for permissible occupational and/or nonoccupational exposure of humans to electromagnetic fields. The standards are expressed as maximum power densities or field intensities in specific frequency ranges and for indicated exposure durations.

The present U.S. standard is based on average power densities and is essentially the same as the American National Standards Institute Radiation Protection Guide ANSI C95.1 (ANSI, 1974). Under this standard, which applies to the frequency range from 10 MHz to 100 GHz, none of the following values, when averaged over any exposure period of 0.1 hour, should be exceeded:

- Power density: 10 $\mathrm{mW/cm^2}$
- Square of electric field strength (E^2_{rms}): 40,000 $\mathrm{V^2/m^2}$
- Square of magnetic field strength (H^2_{rms}): 0.25 $\mathrm{A^2/m^2}$
- Energy density: 1 $\mathrm{mWh/cm^2}$.

The values of E^2_{rms} and H^2_{rms} above are approximately the "free-space equivalents" of 10 $\mathrm{mW/cm^2}$ (100 $\mathrm{W/m^2}$) power density--i.e.,

$$E^2_{rms} = (Z) \times (100\ \mathrm{W/m^2}) \quad (1)$$

$$H^2_{rms} = (1/Z) \times (100\ \mathrm{W/m^2}) \quad (2)$$

where Z represents the "impedance" or value of E/H for free space, but rounded off from 377 ohms to 400 ohms to yield E_{rms} = 200 V/m and H_{rms} = 0.5 A/m instead of values to more than one significant figure. Where only one type of field is present, the appropriate value of maximum field applies. Where both types of field are present (e.g., from separate sources of magnetic and electric fields), the maximum energy-density value above represents an additional condition that the total contributions from both fields, averaged over any 0.1-hour period, should not exceed the equivalent of 10 $\mathrm{mW/cm^2}$. The standard states that these maximum permissible levels are appropriate for exposure under moderate environmental conditions (temperature and humidity); lower values should be used under environmental conditions that induce significant heat stress.

The 10-mW/cm^2 values originated from (a) the physiological consideration that whole-body exposure of a human to levels from about 100 mW/cm^2 upward would produce a mild to severe increase in thermal load (depending on the level) and (b) the application of a safety factor of 10 to the lower limits of this power-density range.

The U.S. standard does not contain specific enforcement or punitive provisions for violations. It has been promulgated by the Occupational Safety and Health Administration (OSHA) as a radiation protection guide for occupational exposure and has been adopted by a number of organizations, including the Department of Defense. The principle underlying this guideline was the belief, based on the then available scientific evidence, that nearly all workers can be exposed to such a level during the normal series of working days without adverse effects. The guideline thus recognized that electromagnetic fields might cause biological effects that have no medical consequences, or that the workers could readily accommodate to the effects.

Based on recent experimental and theoretical results, the EPA, the NIOSH, and the ANSI are considering possible revisions to the U.S. standard. A provisional frequency-dependent standard based on an average SAR limits of 0.4 W/kg in exposed tissue is under discussion by the ANSI Subcommittee C95.4 to be applicable to both occupational and non-occupational exposure. However, environmental levels of electromagnetic fields are very much lower than occupational levels, and the question of environmental standards for the general (nonoccupational) population is still under consideration by the EPA.

Since other countries may be exposed to the sidelobe RFR from U.S. SPSs, a discussion of the standards of other countries is pertinent.

Present standards in the United Kingdom, France, and West Germany are essentially the same as the current ANSI guideline (Stuchly and Repacholi, 1978). This was formerly true for Canada also. However, the Canadian federal government has recently revised its standard along the lines shown in Table 2-1. The maximum permissible general (nonoccupational) level for continuous exposure is 1 mW/cm^2, applicable to frequencies in the band from 10 MHz to 300 GHz. For occupational

Table 2-1

CANADIAN STANDARD OF MAXIMUM PERMISSIBLE EXPOSURE LEVELS

Exposure	Frequency (GHz)	Maximum Level*	Duration
General public	0.01 to 300	1 mW/cm^2	24 h
Occupational	0.01 to 1	60 V/m (1 mW/cm^2) 0.16 A/m (1 mW/cm^2) 1 mW/cm^2	8 h
		1 to 25 mW/cm^2	t(min) = 60/P P = power density
	1 to 300	5 mW/cm^2	8 h
		1 to 10 mW/cm^2	t(min) = 300/P
		10 to 25 mW/cm^2	t(min) = 60/P

* Values in parentheses are approximate "free-space-equivalent" power densities.

exposure, the maximum levels are frequency- and duration-dependent. For example, for the frequency range from 1 to 300 GHz, the new standard permits exposure to 5 mW/cm^2 for a maximum of 8 hours/day, up to 10 mW/cm^2 for 6 minutes or less, and up to 25 mW/cm^2 for 2.4 minutes or less.

The Swedish standard, which formerly was essentially the same as ANSI C95.1, was revised in 1976 as shown in Table 2-2 (Stuchly and Repacholi, 1978). Again, the new maximum occupational exposure levels are about tenfold lower than they were. The new standard is assumed to apply to the general (nonoccupational) population as well.

Presumably, the reductions of maximum permissible levels in the Canadian and Swedish standards were engendered in part by consideration of some of the relatively recent research results indicative of bioeffects due to chronic exposure at power densities in the range from 1 to 10 mW/cm^2. For similar reasons, it is likely that the U.S. standard may also be comparably reduced.

In the USSR, the maximum level for 24-hour exposure of the general population is 5 $\mu W/cm^2$ (Shandala, 1978; McRee, 1979). The occupational standard is summarized in Table 2-3 (Stuchly and Repacholi, 1978; McRee, 1979). It specifies higher maximum levels than for the general population. For example, in the frequency range from 300 MHz to 300 GHz, it permits levels from 10 $\mu W/cm^2$ for a full working day to 1 mW/cm^2 for 20 minutes of exposure. However, the Soviet military services and establishments are specifically exempted from such standards. The process by which the USSR standard was arrived at is unknown, because the Council of Ministers that sets standards does not publish its proceedings. Nevertheless, we can surmise that the standard is based in part on the claimed existence of "nonthermal" effects and on the philosophy that exposure to any power-density level is potentially harmful, leading to the application of large safety factors in formulating maximum permissible levels (but exempting the military from such standards, as mentioned above).

Table 2-2

SWEDISH OCCUPATIONAL STANDARD OF MAXIMUM PERMISSIBLE EXPOSURE LEVELS

Frequency (GHz)	Maximum Power Density (mW/cm^2)	Exposure Duration (h)	Remarks
0.01 to 0.3	5	8	Averaged over 6 min
0.3 to 300	1	8	Averaged over 6 min
0.01 to 300	25		Averaged over 1 s

Table 2-3

USSR MAXIMUM PERMISSIBLE LEVELS FOR OCCUPATIONAL EXPOSURE

Frequency (GHz)	Exposure Limit*	Exposure Duration	Remarks
0.01 to 0.03	20 V/m (100 $\mu W/cm^2$)	Working day	--
0.03 to 0.05	10 V/m (25 $\mu W/cm^2$), 0.3 A/m (4 mW/cm^2)	Working day	--
0.05 to 0.3	5 V/m (6 $\mu W/cm^2$)	Working day	--
0.3 to 300	10 $\mu W/cm^2$	Working day	Stationary antennas
	100 $\mu W/cm^2$	Working day	Rotating antennas
	100 $\mu W/cm^2$	2 h	Stationary antennas
	1 mW/cm^2	2 h	Rotating antennas
	1 mW/cm^2	20 min	Stationary antennas

---

[*] Values in parentheses are approximate "free-space-equivalent" power densities.

3 ANALYSIS OF SCIENTIFIC INFORMATION

In an environmental effects assessment, the interpretation of scientific information presents some novel problems that lie outside of the ordinary realm of scientific investigation. In traditional scientific procedure, a statement or proposition is considered true if--and only if--it can be proven by objective evidence and can be related logically and theoretically to the existing body of scientific knowledge. Speculation about what else might be true is appropriate as the next step in the advancement of scientific knowledge, but in traditional science, speculation is clearly distinguished from established fact and usable theory. Expansion of scientific knowledge ordinarily comes from investigation of questions suggested by the results of past (usually recent) discovery; hence the direction of scientific investigation is usually controlled by the results achieved, rather than by specific information desired.

An environmental effects assessment, or any assessment of potential technological hazards to mankind, departs from traditional scientific procedure in two significant ways. First, the direction of investigation is controlled by the need for specific kinds of information, often formulated by agencies that do not conduct the investigation. Second, the investigation is often directed at establishing the absence of harmful or deleterious effects. The first departure sometimes results in the inefficient use of scientific manpower and resources: the second requires proving a negative statement, which is methodologically impossible. The only feasible accommodation to proving the absence of hazardous effects is to establish a battery of standardized laboratory tests to which an agent or chemical must be submitted, and to designate it as hazardous if it shows effects in any of the tests. Proper application of such a principle also requires that the benefits and costs of the test system must be weighed against the benefits and costs of the environmental agent in question.

The foregoing problems apply to any kind of risk assessment, whether it be for the Food and Drug Administration, the Environmental Protection Agency, or a private insurance company. In the case of this survey on the environmental assessment of effects of radiofrequency radiation, the historical development of a field of study has incorporated two problems that seem to be peculiar to this type of environmental agent. These problems are considered briefly in the following paragraphs.

The first problem involves the long-standing debate over thermal versus nonthermal effects. The technical aspects of the question of thermal versus nonthermal effects is discussed in some detail in Section 5 of this document. What is discussed here is the philosophical aspect of the problem as it relates to the evaluation of the available scientific information. Based on review of the literature, it seems that the problem constitutes a misdirection of scientific effort. Because most of the absorbed energy of radiofrequency radiation is deposited in the form of heat, it is virtually impossible to prove that a given effect is "nonthermal" even though it is obtained at a power-density level that appears to be too low to induce a rise in temperature in the irradiated subject. Even the absence of the effect when thermal load is induced by other agents (i.e., hot air) does not prove the nonthermal nature of the effect, since the rate of absorption and distribution of the thermal load will differ from those induced by radiofrequency radiation. In this assessment, the question of thermal versus nonthermal as a basis for the mechanism of an effect is largely ignored, and the attention is directed instead to the power-density level and duration or repetition of exposure that produce or fail to produce the effect.

The second problem involves the question of the thousandfold difference between the USA and the USSR in standards for permissible occupational exposure to microwave irradiation. This difference is often considered to be evidence for (unspecified) deleterious effects of microwaves that officials in the USA have failed to recognize. However, occupational or environmental standards for exposure to any agent involve at least two independent factors: 1) the scientific information available for making a decision and 2) the regulatory philosophy on which the bureau charged with setting standards operates. With regard to the first factor, all scientific information available to the preparers of this assessment has been reviewed carefully by reasonably strict standards of scientific competence. With regard to the second factor, there is a profound difference in regulatory philosophy between the two countries (Zielhuis, 1974). In the USA, effects are usually recognized as hazardous in terms of their frank pathological nature or the discomfort experienced by exposed subjects. In the USSR, any effect resulting in a change in any physiological function by more than two standard deviations from the norm in any individual is regarded as unacceptable. It is inappropriate to discuss the merits of the two philosophies in detail, but two points in the philosophical position of the USSR may be noted. First, biochemical and physiological norms of the human population rarely have perfect Gaussian distributions with well-defined standard deviations; hence, application of the criterion of hazard becomes difficult. Second, human experience shows that there are few substances or agents that have no significant effect on the entire population. Even natural foods that man has eaten for thousands of years may provoke violent allergic reactions in a few individuals. Hence, any criterion of absolute safety is illusory.

In developing the assessment that follows, the main areas of concern have been the credibility and reliability of the scientific data available for review and the information on physiological mechanisms, dose-response curves, and power-density or dose thresholds. Attention is also given to the question of whether specific effects may be relevant to human safety and health, but these are subjective matters, and othersmight disagree with the assessment. Finally, speculation about what effects *might* occur is restricted to narrow areas of physiology where there are well-defined mechanisms that could lead to effects that have not yet been observed or recorded.

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4 OTHER REVIEWS

This section contains descriptions of representative general reviews of the literature on the bioeffects of RFR, including two by Eastern European authors (Baranski and Czerski, 1976; Sudakov and Antimoniy, 1973) and two of Eastern European research by an American (McRee, 1979, 1980). The bibliographies in these reviews served as additional sources of possibly relevant literature citations, thereby ensuring adequate coverage of the literature. Although the conclusions and opinions of the authors of these reviews were carefully examined, the conclusions stated in this assessment of the SPS were derived independently by analyses of the primary research literature on each bioeffects topic.

Two useful recent compendia are the issue of the *Bulletin of the New York Academy of Medicine* (1979) that covers the "Symposium on Health Aspects of Nonionizing Radiation" held at the Academy in April 1979 and the *Proceedings of the IEEE*, *Special Issue on Biological Effects and Medical Applications of Electromagnetic Energy* (1980). Both publications contain reviews of specific RFR bioeffects topics as well as reviews of the entire field. The presentations in the *Bulletin* are directed primarily toward acquainting physicians about the status of the field, whereas those in the *Proceedings* are primarily for the nonspecialist. In addition, the *Proceedings* contains first publication of some recent research results. Only the general review articles in these issues and those selected from earlier publications are considered in this section.

In the *Proceedings*, Michaelson (1980) presents an overview that includes brief discussions of principles of biological experimentation and interpretation of results, the necessity for and the problems associated with scaling and extrapolating results on animals to humans, and some basic physiological considerations involved in exposure to RFR. He then summarizes the current state of research on all of the major bioeffects topics. He concludes that most of the experimental data indicate that the reported effects of RFR exposure are primarily due to temperature increases or internal changes in temperature gradients, but recommends further research in areas where there are substantial uncertainties and disagreements, particularly on the effects of chronic exposure to low levels of RFR. Michaelson cites 99 references. An earlier review by Michaelson (1978) covers much of the same subject matter and provides 209 reference citations.

In both the Proceedings and the Bulletin, McRee (1979, 1980) reviews the difficulties in assessing the Eastern European literature on bioeffects of RFR prior to about 1972, and he discusses the inception of the cooperative agreements between the USSR and the USA. He also indicates that the initial stages of the cooperative program primarily involved exchanges of information (and interchange visits). It became evident that most of the USSR research involved chronic exposures to average power densities of about 500 μW/cm^2 and lower, whereas the U.S. research involved relatively short exposures to about 5 mW/cm^2 and higher. This situation led to an agreement to perform duplicate experiments in the two countries. In the duplicate experiment that McRee describes, rats were exposed at 500 μW/cm^2 for 7 hours/day, 7 days/week for 3 months, and specific behavioral and biochemical tests were performed. The U.S. study found a decrease in sulfhydryl activity and blood cholinesterase, as did the USSR study, and blood chemical analyses at the end of the 3-month exposure period showed aldosteronism in the exposed animals, relative to controls, due to vacuolated and hypertrophied zona glomerulosa of the adrenal glands. Also, significant differences in the same direction as the USSR results were found in all behavioral parameters studied (increased threshold in footshock detection, decreased activity in an open field, and poor retention of an avoidance response). In both reviews, McRee also summarizes effects on humans and animals reported by Soviet, Polish, and Czechoslovakian scientists, and he discusses the safety standards in these and Western countries in the Bulletin. McRee cites 33 references in the Proceedings and 5 references in the Bulletin.

In the Bulletin, Cleary (1979) presents a brief overview, with emphasis on reported effects of exposure at low average power densities. He indicates the difficulties in making quantitative comparisons of results and extrapolating from data on animals to effects on humans. He cites 22 references. In a more comprehensive, earlier review (Cleary, 1977), he analyzes the results of 12 studies on various aspects of RFR bioeffects and includes references to 100 other articles. He discusses the physical characteristics of RFR, the mechanisms of interaction of RFR with biological systems, and whole-body dose rates and dose-rate distributions within actual and model biological systems. He then reviews the major physiological and behavioral effects of RFR.

Carpenter (1977) gives a critical, comprehensive presentation of RFR and its effects, emphasizing RFR as an environmental agent. Sections deal with physical characteristics and properties of RFR, effects on tissue, "thermal" and "nonthermal" effects, exposure levels, biological effects of RFR on human beings and experimental animals, and RFR effects on the eye, the testes, the nervous system, and on development. Carpenter cites 110 references.

Two reviews, one covering RFR biophysics and the other discussing biological and pathophysiological effects of exposure to RFR, are contained in the transactions of a short course held in Ottawa, Canada, in June 1978. Lin (1978) presents an assessment of the current knowledge about RFR interactions with biological systems, with emphasis on the dielectric properties of tissue materials, propagation and absorption of RFR in tissues, and basic physical mechanisms of interaction. He cites 76 references.

Stuchly (1977) reviews potentially hazardous RFR sources, citing 38 references. The review discusses those sources judged to have potential for producing hazardous levels of RFR under normal operating conditions and under possible malfunction, and considers satellite communications systems and microwave-power devices for generating heat.

Dodge and Glaser (1977) assess international trends in research, development, and occupational health and safety, concentrating on events since 1975. Some 25 references are cited. Sections discuss exposure standards, research on bioeffects, effects of RFR on humans, and U.S. federal RFR health and safety programs.

A very comprehensive Eastern European presentation of the then current literature and research results is the 234-page book by Baranski and Czerski (1976) published in English (translation by Czerski). The book contains references to 614 articles, with wide representation given to Western, as well as Eastern European, investigations. The seven chapter headings are:

Introduction
Physical Characteristics of Microwaves
Interaction of Microwaves with Living Systems
Biological Effects of Microwaves. Experimental Data
Health Status of Personnel Occupationally Exposed to Microwaves, Symptoms of Microwave Overexposure
Safe Exposure Limits and Prevention of Health Hazards
Final Comments

Sudakov and Antimoniy (1973) provide an extensive review (224 references) of the neurophysiology and behavior of animals and humans, in an English translation of the original Russian article by the Joint Publications Research Service. The authors appear to accept as uncontestable the premise that RFR has direct effects (denoted by them as "nonthermal") on the nervous system of animals. The review is in two main sections. The first is concerned with biological aspects of the effects of RFR on the central nervous system (CNS) of animals and man; it contains subsections on natural RFR as a factor in evolution, the sensing of RFR by living organisms, and the effects of natural RFR on animals and man, on the activity of the CNS, and on the behavior and conditioned activity of animals and man. The second main section is concerned with neurophysiological mechanisms of action of RFR, with subsections on bioelectrical activity of the brain during exposure to RFR, morphological and functional changes in the CNS on exposure to RFR, and selective action of RFR on structures of the CNS.

5 PRESENT STATE OF KNOWLEDGE REGARDING PHYSICAL EFFECTS

5.1 INTERACTIONS OF RFR WITH BIOLOGICAL ENTITIES

Because of the frequent usage, in the literature, of "thermal" and "nonthermal" to characterize interactions of RFR with biological entities and the controversy and confusion engendered by such usage, it is appropriate to introduce working definitions of these terms, with the recognition that the boundary between these types of interactions is not sharp.

The interaction of RFR with a biological entity can be characterized as thermal if the energy absorbed by the entity is transformed into heat at the absorption site. Implicit in this definition is that "heat" is a macroscopic quantity involving many molecules. Heat absorption, in turn, is defined in classical thermodynamics as either an increase in the mean random speed (or kinetic energy) of the molecules at the absorption site (a local increase in temperature) or an increase in the disorder or randomness of the molecular motion at the site without an increase in temperature (a first-order phase change), or both.

Energy can also be absorbed by an entity at specific discrete frequencies in the form of energy packets or "quanta," each of which has an energy proportional to one of the discrete frequencies. Although large numbers of molecules can be involved, quantum absorption is essentially a microscopic phenomenon in that the constituents and configurations of the various molecular species composing the entity determine the specific frequencies or characteristic spectra at which quantum absorption can occur. The kinds of interactions involved are numerous and of varying degrees of complexity. They include alterations of molecular orientations and configurations that do not change the basic identities of the molecules, disruption of intermolecular or intramolecular bonds, and excitation of atoms or molecules to higher electron states (including ionization). Such interactions can be characterized as "short-range" processes. There are also cooperative interactions among subunits of molecules within biological cells, in cell membranes, and in extracellular fluids. Cooperative interactions are often characterized as "long-range" because absorption of energy at one specific site in a structure (e.g., in a membrane or in a biological macromolecule) can affect a process elsewhere in the structure, or a function of the structure as a whole can be triggered by the release of energy stored in the structure, thereby producing biological amplification.

Conceptually, all such quantum interactions can be characterized as "nonthermal." However, if most of the energy thus absorbed is subsequently transformed locally into heat (as defined above), the distinction between nonthermal and thermal is blurred. Pragmatically, therefore, characterization of an interaction of RFR with a biological entity as nonthermal requires that the interaction give rise to a frequency-specific effect that is experimentally distinguishable from heating effects due to thermalization of the absorbed RFR energy.

5.1.1 Thermal Interactions

Consider now the incidence of continuous-wave (CW) RFR on a human or an animal. The relative magnetic permeability of most organic constituents is about unity. Therefore, thermal interactions (as defined above) can be described primarily in terms of the dielectric, electrical-conductivity, and thermal properties of the body organs, tissues, fluids, and so forth, as well as the characteristics of the RFR (frequency, power density, polarization). These properties have been measured for various mammalian tissues, blood, cellular suspensions, protein molecules, and bacteria over the spectral region from about 10 Hz to 20 GHz, notably by Schwan and coworkers (Schwan and Foster, 1980; Schwan, 1963, 1957; Schwan and Piersol, 1955; Schwan and Li, 1953), as well as others (Lin 1975; Cook, 1951, 1952). In general, the dielectric constants were found to vary with frequency in a separate characteristic manner for each of three parts of that frequency range ("alpha," "beta," and "gamma" dispersion regions), as shown for muscle tissue in Figure 5-1. These dispersion regions are ascribed to different predominant relaxation mechanisms, each characterized by specific time constants (Schwan, 1957). In the low and intermediate frequency ranges (about 10 Hz to about 100 MHz), encompassing the "alpha- and beta-dispersion" regions, the properties of cell membranes, which have large specific capacitances (about 1 microfarad/cm^2), predominate. In the range above about 10 GHz ("gamma-dispersion" region), membrane impedances are negligible and the behavior of the water and electrolyte content are most predominant.

In the frequency range from about 300 MHz to about 10 GHz, the dielectric constants of skin, muscle, and blood vary relatively little with frequency because the transition between the beta- and gamma-dispersions occurs in this range. (This behavior is illustrated in Figure 5-2 for muscle.) The mean dielectric constants for these three constituents are about 40, 50, and 60, respectively; the differences in values are largely ascribable to the proportion of water in each constituent, water having a dielectric constant of about 80.

Because the index of refraction of any material is related to its dielectric constant, electromagnetic fields are reflected and refracted at the air-surface interface and at internal boundaries between constituents of widely different dielectric properties (e.g., at interfaces between the skull and the dura or between a body cavity and adjacent tissues), thereby affecting the internal field distributions. Figure 5-3 displays plots of the power transmission factor at air-muscle, fat-muscle, and air-fat interfaces over the frequency range from 100 MHz to 10 GHz. It is seen that at an air-muscle interface, for example, only about 44% of the incident power density of 2.45 GHz RFR is transmitted (the remainder being reflected). The corresponding value for the air-skin interface is approximately the same. The fraction entering a body is progressively attenuated with depth because of energy absorption.

The attenuation constant (rate of energy absorption with distance) of any material is proportional to the square root of its electrical conductivity. The concept of "penetration depth" (inverse

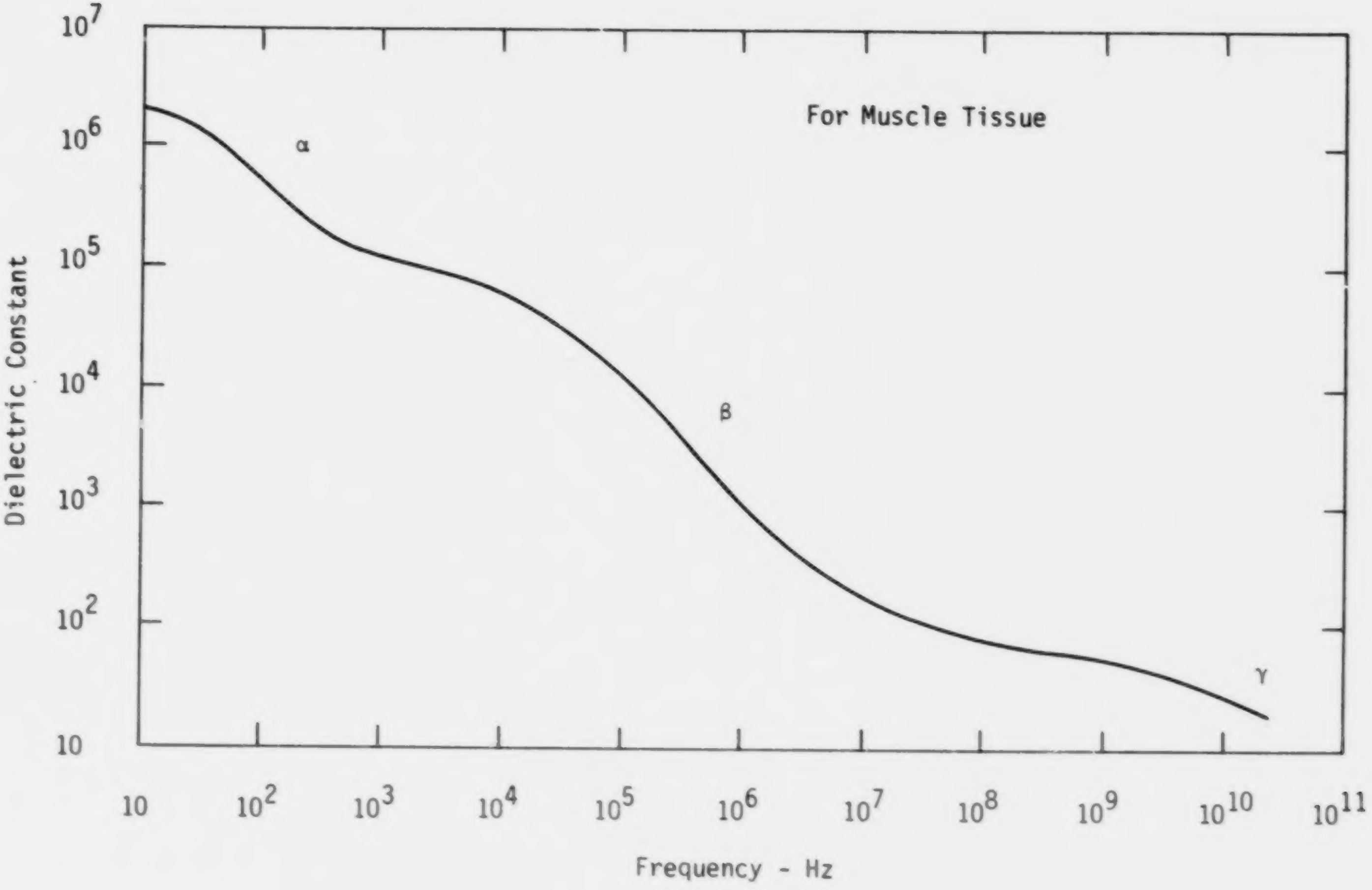

FIGURE 5-1 DIELECTRIC DISPERSION FOR MUSCLE TISSUE

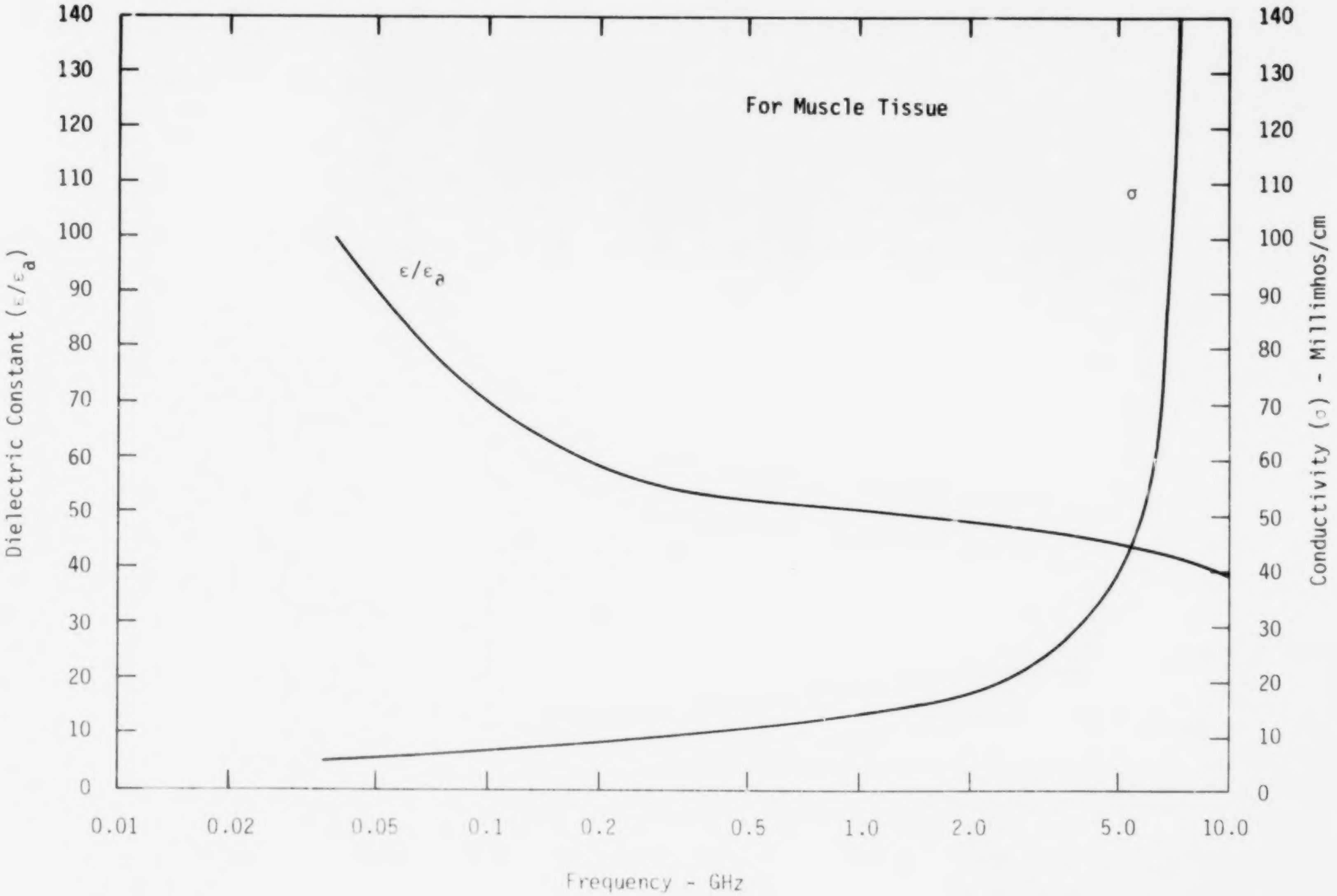

FIGURE 5-2 VARIATION OF DIELECTRIC CONSTANT AND ELECTRICAL CONDUCTIVITY WITH FREQUENCY FOR MUSCLE TISSUE

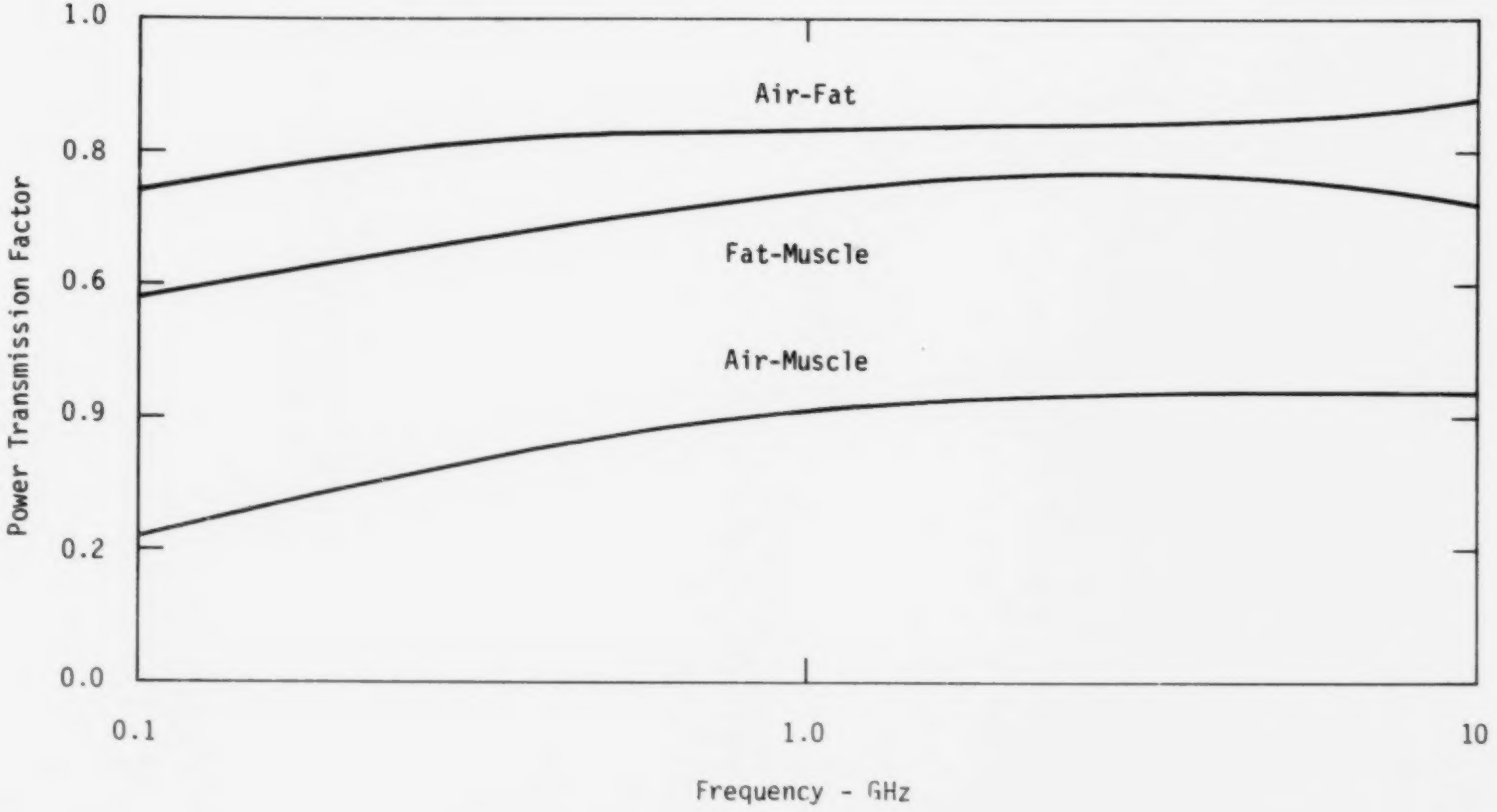

FIGURE 5-3 POWER TRANSMISSION FACTORS AT SEVERAL INTERFACES

of attenuation constant) is often used. For homogeneous specimens, the penetration depth is defined as the distance at which the electric field amplitude is 1/e (37%) of its value or the power density is $1/e^2$ (14%) of its value just within the surface. The electrical conductivities of skin, muscle, blood, and other constituents of the body increase slowly with frequency up to about 1 GHz and rapidly from about 1 GHz upward. (See, for example, Figure 5-2.) The penetration depths for these three constituents are shown in Figure 5-4. At 2.45 GHz, for example, the penetration depth for muscle is about 1-3/4 cm, whereas at about 10 GHz and higher, field penetration is confined to the skin.

5.1.2 Dose-Rate Considerations

In the literature on bioeffects of RFR, thermal energy absorption from an electromagnetic field is usually characterized by the Specific Absorption Rate (SAR), defined as the rate of energy absorption in a small volume at any locale within an entity, divided by the mean density of the constituents in that volume. SAR is expressed in terms of W/kg or mW/g. The numerical value of SAR in any small region with a biological entity depends on the characteristics of the incident field (power density, frequency, polarization) as well as on the properties of the entity and the location of the region. For biological entities that have complex shapes and internal distributions of constituents, spatial variations of SAR are not readily calculated. Therefore, the concept of "mean SAR," which represents the spatial average value for the body per unit of incident power density, is often used because it is a quantity that can be measured experimentally (e.g., by calorimetry) without requiring information on the internal SAR distribution.

Many investigators have studied relatively simple geometric models, including homogeneous and multilayered spheroids, ellipsoids, and cylinders that have weights and dimensions approximately representative of various species, including humans. Such models were actually--or were assumed to be--irradiated with linearly polarized

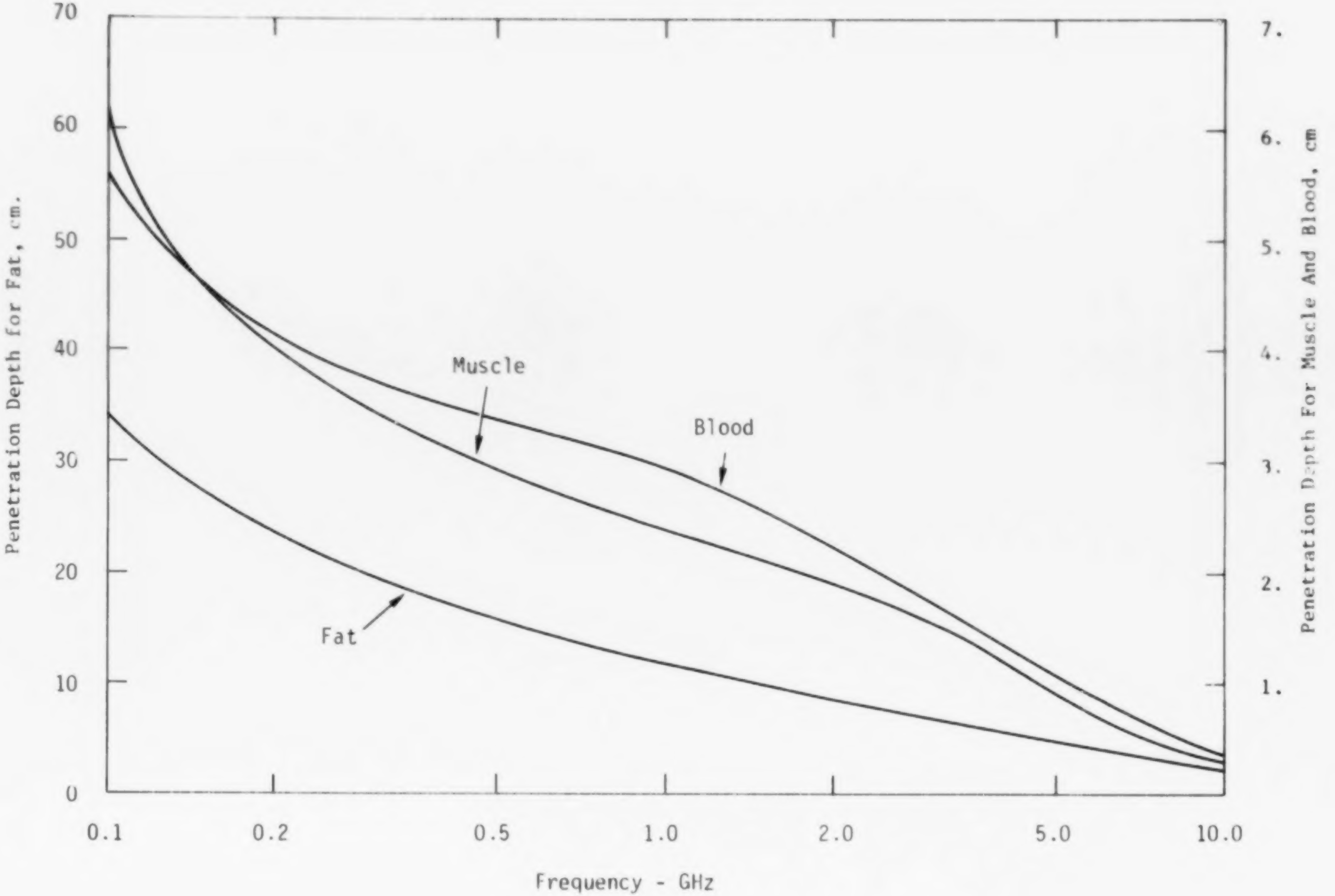

FIGURE 5-4 PENETRATION DEPTH VERSUS FREQUENCY

plane waves to determine the dependence of mean SAR on frequency and orientation of the object relative to the polarization direction of the RFR. Many of the significant data have been included in a compendium (Durney et al., 1979, 1978) that is useful for very approximate frequency-scaling and interspecies comparisons of mean SAR values. An important result of this work is that the largest value of mean SAR is obtained when the longest dimension of each kind of model is parallel to the electric component of the field and when the wavelength of the incident RFR is about 2.5 times the longest dimension. The adjective "resonant" is often applied to the frequency corresponding to this wavelength. The resonant value of mean SAR for each model is also inversely dependent on the dimension perpendicular to the polarization direction (and propagation direction) of the field--i.e., the model has characteristics somewhat similar to those of a lossy dipole antenna in free space. Resonances would also occur for circularly polarized RFR. Such RFR can be resolved into two mutually perpendicular components, each having half the total power density. Therefore, an entity exposed to circularly polarized RFR would have lower mean SAR values at resonance than it would have if exposed to linearly polarized RFR of the same total power density.

Figure 5-5 shows plots of mean SAR versus frequency for a prolate-spheroidal model of an "average" man, approximately 5 ft 9 in. (1.75 m) tall and weighing about 154 lb (70 kg), exposed to 1 mW/cm^2 of plane-polarized RFR in three orientations relative to the polarization direction. (A relatively sharp peak is obtained at resonance for the "E" orientation) in which the long axis of the prolate spheroid is parallel to the polarization direction (electric vector) and perpendicular to the magnetic vector and propagation direction. (In the "H" orientation, the long axis is parallel to the magnetic vector and perpendicular to the electric vector and propagation direction; in the "K" orientation, the long axis is parallel to the propagation direction.) It is seen that for this model of man, the resonant frequency (in the E orientation) is about 70 MHz; at this frequency the mean SAR is about 0.2 W/kg for 1 mW/cm^2 incident power density, or about 1/6 of his resting metabolic

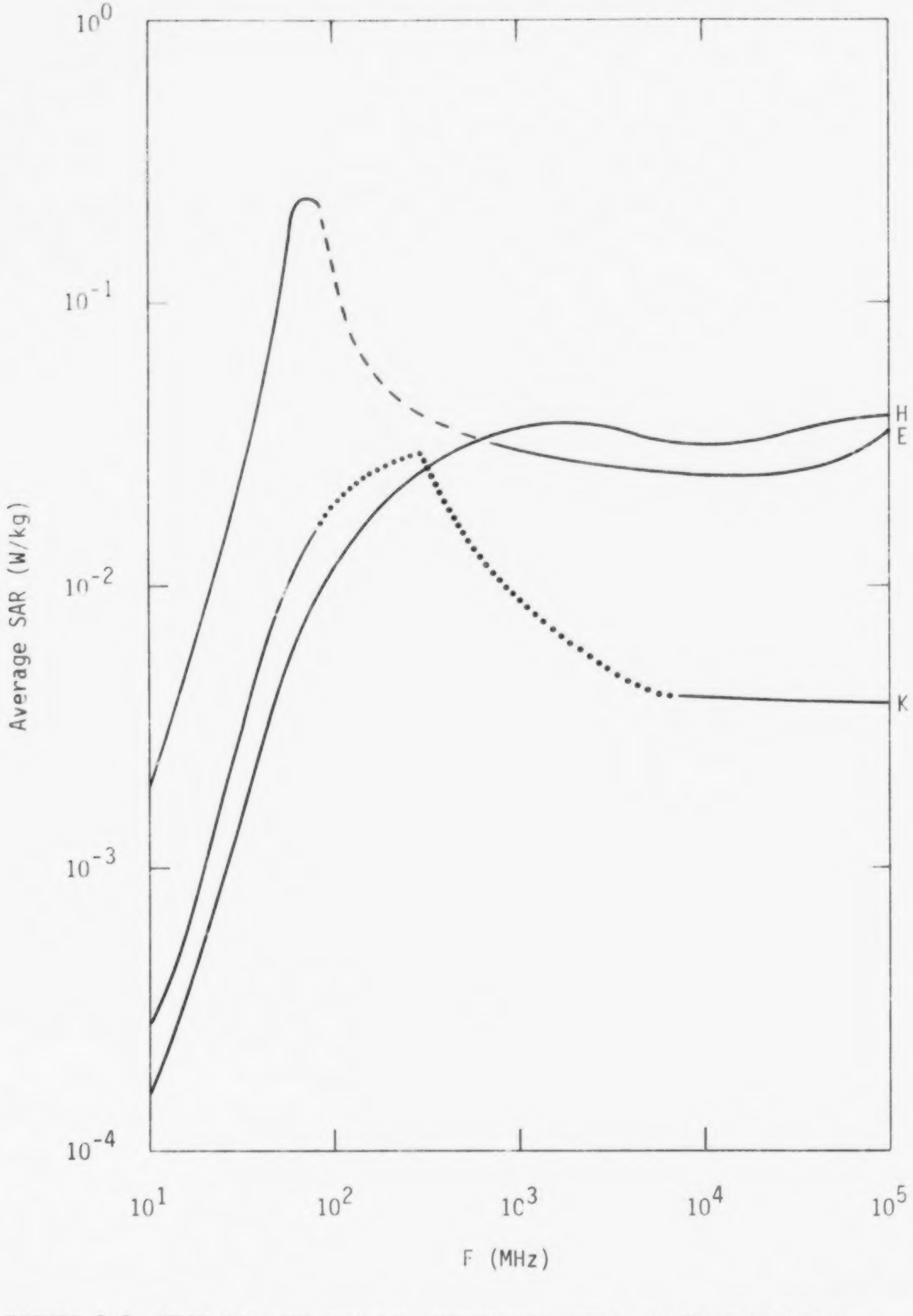

FIGURE 5-5 MEAN SAR FOR PROLATE-SPHEROIDAL MODEL OF "AVERAGE" MAN EXPOSED TO 1 mW/cm^2 OF RFR

exercise ranging from walking to sprinting (Ruch and Patton, 1973). Similarly, the resonant frequency for an "average" woman about 5 ft 3 in. tall is about 80 MHz, and her mean SAR is about the same as for the average man. The resonant frequency for a 10-year-old is about 95 MHz; for a 5-year-old, about 110 MHz; and for a 1-year-old, about 190 MHz. The resonant mean SAR values for such children are about 0.3 W/kg for 1 mW/cm^2. The presence of a ground plane or other reflecting surfaces shifts the resonant frequencies downward and can produce higher values of mean SAR at the lower resonant frequencies (Hagmann and Gandhi et al., 1979; Gandhi et al., 1977; Gandhi, 1975).

Below resonance in the E orientation, the mean SAR is approximately proportional to f^2; above resonance, the mean SAR is approximately proportional to 1/f for about one decade of frequency and then it levels off. (These relationships of mean SAR to frequency constitute the basis for the physical aspects of the Exposure Protection Guide currently being considered by the ANSI C95.4 committee.)

Figure 5-6 presents similar data for a prolate-spheroidal model of a "medium" rat (0.2 m long and weighing 0.32 kg). It is seen that not only is the resonant frequency (approximately 650 MHz) higher than the values for humans, but also the resonant mean SAR is larger (about 0.8 W/kg for the rat, versus about 0.2 for man, per mW/cm^2 of incident power density). Therefore, scaling of data from experimental animals to humans must also consider such differences of mean SAR as well as frequency.

The foregoing discussion of mean SAR is also largely applicable to modulated RFR (including pulsed RFR) at corresponding carrier frequencies and time-averaged incident power densities.

To illustrate how the concept of mean SAR could be interpreted, consider the model man. Absorption of energy as heat by exposure of such a model man at his resonant frequency (70 MHz) in the E orientation to an average power density of 1 mW/cm^2 (SAR of 0.2 W/kg) for 1 hour would produce a mean body temperature rise of only about 0.2°C if no heat removal mechanisms are present and if no first-order phase changes are involved. At 2.45 GHz, the mean SAR is about 0.04 W/kg per mW/cm^2

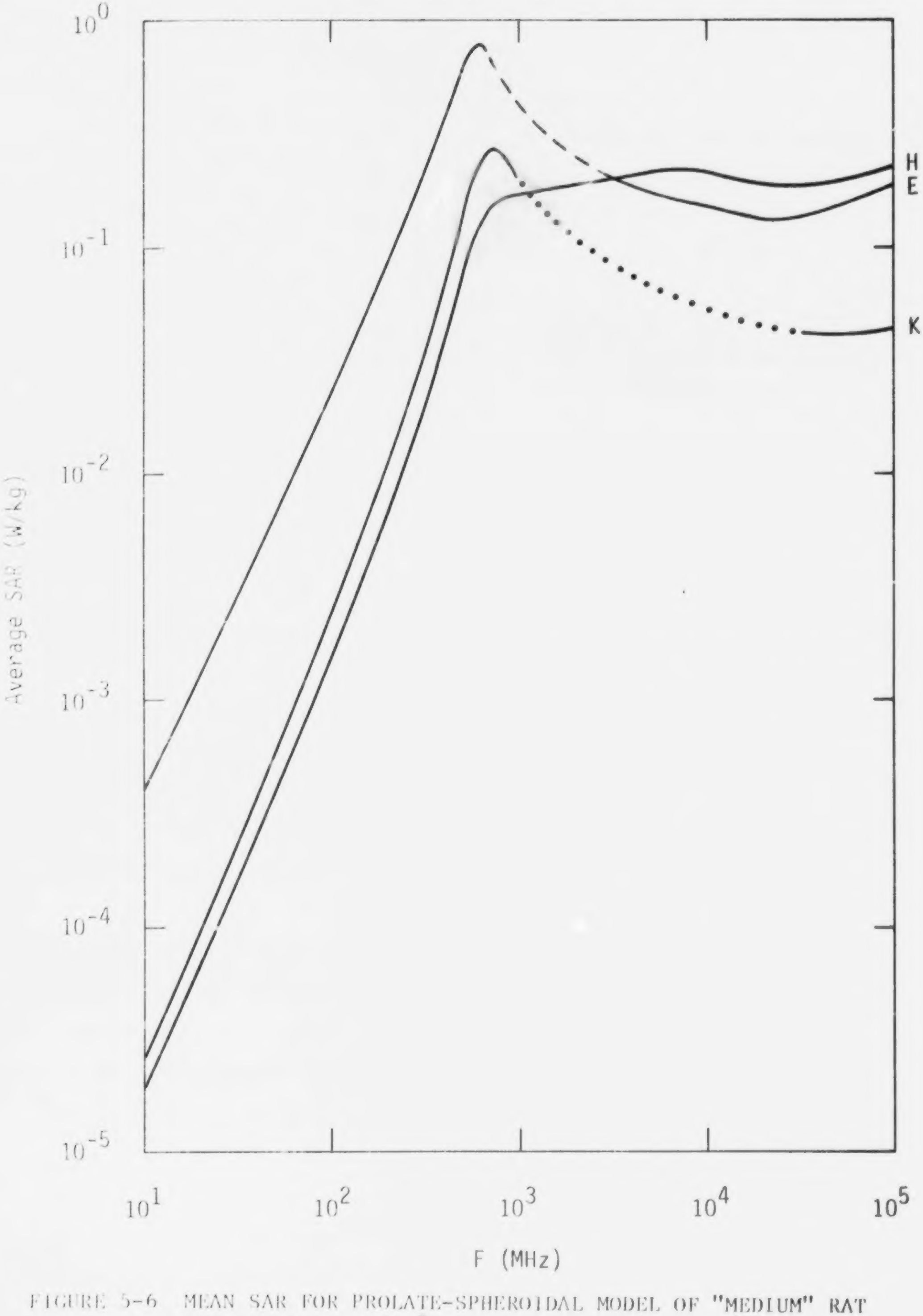

FIGURE 5-6 MEAN SAR FOR PROLATE-SPHEROIDAL MODEL OF "MEDIUM" RAT EXPOSED TO 1 mW/cm^2 of RFR

for the H orientation and about 0.03 W/kg per mW/cm^2 for the E orientation, as seen in Figure 5-5. Therefore, exposure of the model man to the same power density for the same duration, but at 2.45 GHz, would produce mean temperature rises of only about 0.04°C or 0.03°C for the H and E orientations, respectively. By contrast, exposure of the model of the medium rat to 2.45 GHz in either orientation (SAR of about 0.2 W/kg per mW/cm^2, as seen in Figure 5-6) for 1 hour at 1 mW/cm^2 would yield a mean body temperature rise of about 0.2°C, which is about fivefold larger than for the model man at this frequency.

Numerical calculations of internal spatial distributions of SAR have been performed on "block" models, in which the shape of the body is approximated by an appropriate arrangement of many cubical cells, with each cell assumed to be biologically homogeneous and to have constant internal field over its volume when the model is exposed to RFR (Hagmann et al., 1977). Block models, as well as homogeneous and multilayered spheroidal and cylindrical models having appropriate electromagnetic and thermal characteristics, have also been used to represent various parts of the body, such as the head and limbs (Gandhi et al., 1979; Massoudi et al., 1979; Rukspollmuang and Chen, 1979; Wu and Lin, 1977; Neuder et al., 1976; Kritikos and Schwan, 1975, 1976; Lin, 1975; Weil, 1975; Joines and Spiegel, 1974).

Probably the most significant finding for spherical models of the isolated head assumed to be exposed to plane-wave RFR has been the discovery of local regions of relative maximum SAR values. The locations of such regions depend on the size of the head, the electromagentic characteristics of its layers, and the wavelength of the incident field. These regions have been conveniently dubbed "hot spots," even for combinations of incident power density and exposure duration that would produce biologically insignificant temperature rises at such spots. An analysis of a homogeneous lossy spherical head model (Kritikos and Schwan, 1975) indicates that hot spots are inside spheres having radii between 0.1 and 8 cm, and in the frequency range from about 300 MHz to 12 GHz; for larger radii and other frequencies, there are internal hot spots, but the hottest spots are at the front surface (facing the RFR source). Similar results were obtained for multilayered spherical models (Kritikos and Schwan, 1976; Weil, 1975). Specifically, Kritikos and Schwan (1976) analyzed two such models, one with a radius of 5 cm and the other, 10 cm. For the 5-cm head, the hot spots are internal over the frequency range from about 400 MHz to 3 GHz. The highest relative maximum SAR occurs near the center of the head at a frequency of about 1 GHz, and has a value of about 9 W/kg for an incident power density of 1 mW/cm^2. At 3 GHz, the hot spot is at the front surface and has an SAR of about 5 W/kg. (Of course, the mean SARs are considerably lower.) By contrast, for the head of 10-cm radius (about that for an adult human head), there are no deep internal hot spots at any frequency; the hot spots are always at or just beneath the front surface.

Rukspollmuang and Chen (1979), starting with a block model of a multilayered spherical head, found qualitatively similar results. They then studied a block model having a shape and internal structure more closely approximating that of the human head (including eyes, nose, skull bone, and brain) and found that much of the energy within the head would be absorbed by the skull. Specifically, for frontal exposure of this model at 918 MHz (near head resonance), the maximum SAR for the brain region is about about one-third that for the brain region of a 7-cm-radius multilayered spherical model. Also, for frontal exposure of the more accurate model to 2.45 GHz, the induced field is concentrated primarily near the proximal surface and therefore energy dissipation within the brain would be relatively low. Based on these analyses, thermal brain damage is unlikely to occur in the adult human head at 2.45 GHz unless the incident power density and exposure duration are sufficient to heat the entire head to well above normal.

Results of theoretical analyses of simple geometric models have been verified experimentally by constructing physical models from synthetic biological materials that have approximately the same electromagnetic characteristics as their corresponding biological constituents, then exposing such models to sufficient power densities to obtain readily measurable temperature rises, and measuring such rises in temperature immediately after irradiation. Although much useful information has been obtained from models that have relatively simple geometries, human and animal configurations are far more complicated and are different from one another. Therefore, SAR distributions in animal carcasses and figurine-shaped physical models have been determined experimentally (Gandhi et al., 1977; Guy et al., 1976a). Calorimetry has been used to measure whole-body mean SAR values (Allen and Hurt, 1979; Gandhi et al., 1979: Kinn, 1977; Hunt and Phillips, 1972). A widely used technique to determine internal field distribution is to section a carcass or physical model along parting planes of interest, then reassemble and expose it. The spatial temperature distribution over each plane is then measured with scanning infrared thermography immediately after exposure. However, such spatial temperature distributions should not be regarded as the corresponding *in vivo* internal temperature distributions, because the heat-transfer characteristics of such carcasses and physical models are significantly different from those of live animals and do not have the thermoregulatory mechanisms of the latter. Instead, such measured temperature distributions represent approximations to the internal field or SAR distributions.

Among the interesting qualitative results with human figurines is that at frequencies near resonance, the local fields can be much higher for certain regions (such as the neck and groin) than for other body locations. In addition, field distributions for nonprimates are quite different from those for primates; this point should be given proper consideration when the analyst attempts to extrapolate experimental findings on any laboratory animal species to humans, or to compare experimental results on different laboratory species.

5.1.3 Quantum Interactions

Regarding quantum interactions of CW RFR, the activation energies for short-range effects at the molecular level extend from about 0.08 eV (1.3×10^{-20} J) for hydrogen-bond disruption to about 10 eV (1.6×10^{-18} J) for ionization. The corresponding quantum frequencies range from about 19 to 240 THz (1 THz = 10^3 GHz) (Cleary, 1973).

However, an electromagnetic quantum at 1 GHz has an energy of only about 10^{-5} eV (1.6×10^{-24} J) or approximately 1.2×10^{-4} of the energy required for hydrogen-bond disruption, the latter being at the lower end of the energy-activation range cited above. At 100 GHz, the quanta are still more than two orders of magnitude too low to cause hydrogen bond disruption. Therefore, the existence of nonthermal biological effects of CW RFR ascribable to such short-range molecular interaction mechanisms is extremely doubtful.

Biological generation of fields having frequencies in the ELF range (below 100 Hz), such as the electroencephalogram (EEG), is regarded as evidence for the occurrence of cooperative or long-range quantum interactions. Several theoretical models of neuronal membranes (e.g., Schmitt and Samson, 1969; Fröhlich, 1975a, 1975b; Grodsky, 1976) indicate that activation energies or frequencies for cooperative processes can be much lower than those for short-range interactions. The thermal energy corresponding to the physiological temperature 37°C is about 0.027 eV, the spectrum of which encompasses the quantum frequency range for cooperative processes. Consequently, the question has been raised whether postulated effects of weak RFR on cooperative processes, based on theoretical models, would be distinguishable from effects that are spontaneously induced thermally. Alternatively, separation of such RFR interactions from those thermally induced may require that the rates of occurrence of the former exceed the rates for the latter. This requirement implies that for manifestation of such effects of RFR, the intensity of the incident field must exceed minimum values or thresholds related to the specific processes. Because predictions from various theoretical models and related considerations conflict to a significant extent (see Adey and Bawin, 1977; Taylor and Cheung, 1978), the issue of whether weak external fields at frequencies well below the infrared range (i.e., RFR) can alter biological processes is not yet resolved.

5.1.4 Interactions of Modulated RFR

Precise usage of the term CW RFR implies the presence of only a single frequency and of unvarying power density. Although the RFR incident from an SPS at a rectenna site and its surroundings is essentially CW, the presence of the rectenna and reflective objects in its vicinity produces spatial variations of power density. Moreover, a biological entity that moves through this spatially varying field will thereby be exposed to a time-varying or modulated field. Consequently, possible biological effects ascribable to the modulation characteristics per se (as distinguished from the effects of time-averaged power density) must also be considered. The occurrence of such effects would require demodulation and filtering, by the biological entity.

Although postulated nonlinear interaction mechanisms (e.g., Adey, 1980; Adey, 1975b; Adey and Bawin, 1977) are conjectural, in vitro effects ascribed to cooperative processes have been reported, notably field-induced increases and decreases of calcium-ion binding to cell membranes of isolated neonate chick brains, a phenomenon called "calcium efflux" (irrespective of the direction of the change). Specifically, lower calcium efflux was reported for chick-brain hemispheres excised, incubated in physiological solution, and exposed for 20 minutes to fields in the ELF range than for similarly treated but unexposed hemispheres (Sheppard et al., 1979; Blackman et al., 1979; Bawin and Adey, 1976b). Statistically significant results were reported for modulation frequencies from 6 to 20 Hz, with highest response at 16 Hz. This phenomenon was not observed with CW (unmodulated) RFR at 147 MHz (Bawin, Kaczmarek, and Adey, 1975) or with CW (unmodulated) RFR at 450 MHz (Bawin, Sheppard, and Adey, 1978a); however, higher calcium efflux was reported for brain hemispheres exposed to ELF-modulated RFR at these carrier frequencies. The latter effect was observed for incident average power densities within the range from 0.1 to 1 mW/cm^2 but not below or above this range, indicating the existence of a power-density "window." Bawin et al. (1977b) also reported preliminary results of increased calcium efflux from the cerebral cortex of the paralyzed awake cat exposed to 16 Hz-modulated 450 MHz RFR at an incident power density of 375 μW/cm^2.

The likelihood that a biological entity that moves through the rectenna area would engender the modulation frequencies and exposure durations necessary for the calcium efflux phenomenon is negligible.

5.1.5 Interactions of Pulsed RFR

Although the RFR from an SPS is CW (or quasi-CW if the aforementioned spatial variations within and near a rectenna site are considered), the effects of high pulse power densities at low average power densities are discussed herein because they are often cited as being "nonthermal" effects.

The temperature rise of any given region within a biological entity due to the arrival of a single RFR pulse would be small, because of the relatively large thermal time constants of biological materials and the operation of heat-exchange mechanisms. However, if the region contains a boundary between layers of widely different dielectric properties, then the temperature gradient (rate of change of temperature with distance) can be large at such a boundary even though the mean temperature rise of the region is small.

One single-pulse effect known to occur in vivo is the phenomenon of "microwave hearing" (Frey and Coren, 1979; Frey, 1961) or the perception of single or repetitive short pulses of RFR as apparently audible clicks. In human volunteers subjected to pulsed fields at 3 GHz, pulse durations of the order of 10 μs and longer and minimum pulse power densities of 300 mW/cm^2 were needed for perception (Cain and Rissman, 1978). The interaction mechanisms involved are not yet completely understood. However, most of the experimental results tend to support the theory that pulse perception occurs because of transduction of the electromagnetic energy into sound pressure waves in the head and normal detection by the auditory apparatus. In one group of suggested mechanisms, transduction is postulated to occur at a boundary between layers that have widely different dielectric properties (e.g., at the boundary between the skull and the skin or dura). The energy in a pulse arriving at such a boundary is converted into an abrupt increase in momentum that is locally thermalized, producing a negligible volumetric temperature rise but a large temperature gradient across the boundary. Under such conditions, rapid local differential expansion would occur, giving rise to a pressure (sound) wave. This effect is often characterized as nonthermal because the power density averaged over two or more pulses can be miniscule. For example, consider two successive pulses, each 20 μs in duration and having 1 W/cm^2 pulse power density (i.e., values well above the threshold). The time-averaged power density would be proportional to these values but inversely proportional to the time interval between the arrival of the pulses at the perceiver. This interval could be indefinitely long without affecting the perception of each pulse. Therefore, the time-averaged power density has no relevance to perception. Irrespective of how the microwave-hearing phenomenon is characterized, the significant point is that the preponderance of experimental evidence indicates that the pulses are converted into actual sound in the head, rather than received by direct RFR stimulation of the auditory nerves or the brain.

As discussed in Section 6, pulsed RFR has been reported to produce other effects, such as alterations of the blood-brain barrier and behavioral changes. However, neither the auditory effect discussed above nor these other effects are likely to be of concern relative to the SPS.

5.2 INSTRUMENTATION FOR DENSITOMETRY AND DOSIMETRY

Much of the early laboratory research on bioeffects of RFR suffered from lack of adequate instrumentation for measuring incident fields or energy absorption rates (e.g., as internal temperature rises at high incident levels) within biological entities. Moreover, the available instrumentation was often incorrectly used or was the source of significant errors in numerical values or of spurious biological findings (artifacts) traceable to perturbations introduced by the presence of the sensors. For these reasons, many of the early results should be viewed as questionable, at least from a quantitative standpoint. During recent years, however, major advances have been made in instrumentation, both for determining incident-field intensities for biological research and for determining internal energy-absorption rates.

Considering first the instrumentation for determining incident fields, a representative device for measuring average power densities is the commercially available broadband isotropic monitor (Aslan, 1972). Its sensors consist of linear arrays of thermocouple elements, each array comprising a lossy antenna of relatively small length and capable of adequate response over the frequency range from 300 MHz to 18 GHz, for which a calibration curve is provided by the manufacturer. Isotropic response is obtained by incorporation of three mutually perpendicular sensor arrays. To minimize errors in the direct-current output values of the sensor assembly caused by possible induction of spurious RF currents in the lead wires, the wires used are of very high resistivity (about 200 kilohms/M). Also, the sensors are only lightly coupled to the incident field, so that perturbations of the field caused by scattering are minimal. The sensors respond to the mean square of only the electric component of the field. Nevertheless, the use of the instrument for measuring average power densities in the far-field region is fully justified because the ratio of the amplitudes of the electric and magnetic components has essentially the same value (377 ohms, the "impedance" of free space) at all points in that region, and the instrument is calibrated to read total average power density. The most sensitive model of this instrument has a full-scale range of 200 $\mu W/cm^2$.

A more recently developed instrument is the National Bureau of Standards (NBS) Model EDM-2 Electric Energy Density Meter, designed for the 10- to 500- MHz range (Belsher, 1975; Bowman, 1974). Its sensor consists of three mutually perpendicular integral dipole-diodes ("rectennas") that also respond only to the electric component of the field. An 18-inch handle from the sensor contains high-resistivity lead wires to minimize field perturbation and spurious pickup. The most sensitive range of the instrument is 0.003 $\mu J/m^3$ full-scale (equivalent to approximately 176 $\mu W/cm^2$), and its response time (rise time plus fall time) is about 1 ms in this range.

Field survey instruments of this kind have been analyzed for possible sources of error (Wacker and Bowman, 1971). Because of the relatively long response times of such instruments, they cannot be used for measuring the pulse power densities of short pulses. Therefore, in research programs on possible bioeffects of pulsed fields, incident pulse power densities are usually calculated from measurements of average power density and duty cycle (or pulse duration and pulse repetition frequency), made with commonly available and readily calibrated components and instrumentation. The use of sophisticated equipment for directly measuring pulse heights (or instantaneous pulse power densities) at low average power densities in bioeffects research is relatively rare.

Magnetic-field probes have been developed for relatively low frequency ranges, as exemplified by the two devices developed at NBS for near-field measurements in the Industrial, Scientific, and Medical (ISM) bands within the range from 10 to 40 MHz (Greene, 1975). The probes consist of single-turn, balanced-loop antennas of 10-cm and 3.16-cm diameter for the amplitude ranges of 0.5 to 5 A/m and 5 to 50 A/m, respectively. (The free-space equivalent power density is proportional to the square of the amplitude. For example, the power-density equivalents to 0.5 and 5 A/m are approximately 10 mW/cm^2 and 1 W/cm^2, respectively.)

The development of assemblies of electric dipoles and magnetic loops for simultaneously measuring both components in the near field for frequencies below 300 MHz was reported from Poland (Babij and Trzaska, 1976).

Regarding methods for determining whole-body dose rates for biological entities and dose-rate distributions within such entities, calorimetry for the former and scanning infrared thermography for the latter (previously discussed in Section 5.1.2) continue to be important techniques which are applicable primarily to animal carcasses and physical models of various species constructed from synthetic biological materials. It is important to note that temperature distributions measured within a carcass by infrared thermography do not represent the in vivo temperature distributions for that animal; rather, they correspond to the incident internal-field distributions.

Probes have been implanted or inserted to measure local RFR-induced temperature changes or fields within animals during irradiation *in vivo*, often with the introduction of artifacts. However, recent developments of probes have largely diminished the problem of perturbation of the temperature or local field caused by the presence of the sensor and its lead wires. Such developments have also reduced the size of readout errors caused by pickup of the incident field in the lead wires and by the presence of spurious potentials at junctions between sensors and lead wires. The miniaturized isotropic dipole-diode probe developed by Bassen and co-workers (Bassen et al., 1975, 1977), the liquid-crystal/fiber-optic probe developed by Johnson and co-workers (Johnson et al., 1975), and the nonmetallic thermocouple developed by Olsen and Molina (1979) are representative examples of such progress.

Efforts are also being made to reduce errors and artifacts in measurements of biologically generated fields and potentials--such as the EEG and the electrocardiogram (EKG)--in the presence of the incident RFR. Chou and Guy (1979) have developed electrodes that can be implanted in the cortex or subcortex for measuring the EEG during chronic exposure to RFR. These electrodes are made of carbon-loaded teflon that has an electrical conductivity close to that of tissue, and they have been shown to be nonpolar, thereby minimizing field perturbations and spurious local potentials. The electrodes have also been shown to have good tissue compatibility by histological examination after 4 to 6 months of implantation. Several high-resistivity electrodes have also been developed by Tyazhelov et al. (1977).

The use of RFR for imaging internal organs is being developed, notably by Larsen and co-workers. For example, using a pair of waveguide antennas (one for transmitting and the other for receiving) submerged in water, Larsen and Jacobi (1979) were able to obtain images of the internals of an excised canine kidney with a resolution of about 5 mm. The kidney was suspended between the antennas, and the antennas were slowly moved together in a successive line pattern (raster) relative to the kidney by a stable electromechanical scanning system. The frequency used was 3.9 GHz, which corresponds to a wavelength of about 8.5 mm in water. The use of submerged phased-array antennas to decrease the scanning time is currently under development.

6 PRESENT STATE OF KNOWLEDGE REGARDING BIOLOGICAL EFFECTS

6.1 EPIDEMIOLOGY

Several epidemiologic studies have been done to determine whether one or more health-related conditions can be associated statistically with chronic exposure to RFR. Representative examples of such studies are presented below. Although none of these studies involved well-defined RFR characteristics or exposure durations, they do represent recent information on possible effects of human exposure to RFR.

The U.S. Embassy in Moscow has been subjected to RFR since 1953, the year after the U.S. moved its chancery to Chekovsky Street (Pollack, 1979). Prior to 1963, the presence of RFR was detected intermittently during routine surveillances of the building, at which time continuous monitoring of the signals was instituted. A study of the health of U.S. personnel assigned to the Moscow embassy during the period from 1953 to 1976, compared with the health of those assigned to other U.S. Eastern European embassies, was conducted by Lilienfeld et al. (1978). The signal frequencies ranged from 2 to 7 GHz (Pollack, 1979), but the modulation patterns were not specified. The maximum incident average power densities and exposure durations varied with the period: 5 $\mu W/cm^2$ for 9 hours/day from 1953 to May 1975; 15 $\mu W/cm^2$ for 18 hours/day from June 1975 to February 7, 1976; and less than 1 $\mu W/cm^2$ for 18 hours/day thereafter. The highest average power density reported was 18 $\mu W/cm^2$ in one part of the southeast corner of the building, where beams from two sources converged.

After considerable effort spent in tracing employees and dependents, 1,827 employees and 1,228 dependents were identified as having been at the Moscow embassy during the 1953-1976 period. The control population consisted of 2,561 employees and 2,072 dependents assigned to embassies and consulates in Budapest, Leningrad, Prague, Warsaw, Belgrade, Bucharest, Sofia and Zagreb during the same time period. Periodic tests for RFR at the control sites showed only background levels.

Medical records were reviewed for 1,209 Moscow employees and 834 dependents. The corresponding numbers for the control group were 1,882 and 1,507. Health questionnaires were returned by 969 Moscow employees and 1,129 control employees. The number of completed dependent questionnaires is not clearly specified in the report.

The authors of this study recognized and commented on the limitations placed on the study by their inability to acquire complete sets of medical records, death certificates, and returned health questionnaires, and by the imprecision of the classification of the individual employees according to probable extent of radiation exposure. Furthermore, they noted that the highest exposure levels were recorded late in the study and therefore, for the subgroup with the highest exposure, the period of time during which health effects might become apparent was the shortest. They also noted that the size of the study population was insufficient to detect excess risks that were less than twofold for many of the medical conditions studied. However, despite these acknowledged limitations, the authors were able to draw the following conclusions.

There were no discernible differences between the Moscow and control groups in total mortality or mortality from specific causes, nor were there differences in mortality between the Moscow and control groups of dependent children or adults. With the exception of cancer-related deaths among female employee groups (both Moscow and control), mortality rates for both Moscow and control groups were less than for the U.S. population at large. Although the study groups were subject to a large variety of health problems, on the basis of the medical records these problems were shared nearly equally by both Moscow and control groups with two exceptions: the Moscow male employees had a threefold higher rate of acquiring protozoal infections, and both men and women in the Moscow group were found to have slightly higher frequencies of most of the common kinds of health conditions reported. However, the authors could not relate these two exceptions to RFR exposure. From the health questionnaire information, the authors reported that there were some excesses in the Moscow employee groups as compared with the controls: more correctable refractive eye problems, more psoriasis in men and anemia in women, and more frequent cases of depression, irritability, difficulty in concentrating, and memory loss. However, the authors noted that "In view of the possibilities which had been publicized of the increased danger to their health and that of their children, it is not at all surprising that the Moscow group might have had an increase in symptoms such as those reported. However, no relationship was found between the occurrence of these symptoms and exposure to microwaves; in fact, the four symptoms mentioned earlier, which showed the strongest differences between the Moscow and Comparison groups, were all found to have occurred most frequently in the group with the least exposure to microwaves."

For dependents, the authors found no differences between the adult Moscow and control groups. Moscow dependent children had twice as high a frequency of mumps as the control children. The incidence of congenital anomalies occurring in children born after arrival of the parents at the duty station was comparable for the Moscow and control groups.

Finally, the authors summarized as follows: "With very few exceptions, an exhaustive comparison of the health status of the State and non-State Department employees who had served in Moscow with those who had served in other Eastern European posts during the same period of time revealed no differences in health status as indicated by their mortality experience and a variety of morbidity measures. No convincing evidence was discovered that would directly implicate the exposure to microwave radiation experienced by the employees at the Moscow embassy in the causation of any adverse health effects as of the time of this analysis."

Two studies have been made of the possible relationship between the occurrence of Down's syndrome (Mongolism) in Baltimore and presumed exposure of the fathers to RFR from radars during military service (Sigler et al., 1965; Cohen et al., 1977). The first study involved 216 mongoloid children and 216 control children matched for hospital of birth (or at home), sex, date of birth, and maternal age at birth, covering the period from January 1946 to October 1962. The data for this study were derived from Baltimore hospital records and interviews with the parents. These data showed that 63.1% of the case fathers and 56.6% of the control fathers had been in the military, but that 8.7% of the case fathers and only 3.3% of the control fathers had reported close association with radars (both within and outside of military service), a statistically significant difference. The authors concluded that "the only truly puzzling association is the suggested relationship between Mongolism and paternal radar exposure," and that "one can only speculate concerning possible mechanisms, but the association between Mongolism and radar exposure deserves further investigation."

In the second study (Cohen et al., 1977), the data from the first study, denoted as the "Original Series," were examined together with data regarding 128 additional matched pairs, denoted as the "Current Series." More detailed questions about RFR exposure and military service were incorporated in the Current Series questionnaires, and service record information on the fathers was acquired. An attempt was made to acquire similarly detailed data on the fathers of the Original Series. In addition, a chromosome study of the fathers was undertaken to determine whether there was any detectable residual damage in the chromosomes of the peripheral blood. After considering the more detailed exposure information, the following findings were reported for the Current Series: 15.7% of case fathers and 21.3% of control fathers had received radar exposure; combining the probably-exposed with the definitely-exposed groups, the corresponding values were 26.0% and 28.3%. The re-evaluated Original Series values for definitely-exposed fathers were 18.6% for case fathers and 15.2% for controls, and when probably-exposed fathers were added the values were 20.6% and 15.7%.

When the data from the Original Series and from the Current Series were combined, the values for case vs control fathers were 17.4% vs 17.5% for definitely exposed and 22.7% vs 20.6% when "some" exposure was included. None of the foregoing comparisons showed statistically significant differences. The results of the chromosome studies have not been reported yet.

The authors concluded that the Current Series did not confirm the suggestions of the Original Series that there was either an excess of radar exposure or a larger proportion of fathers with military service prior to the conception of the cases of Down's syndrome. The authors note that "in view of the suggestive findings of the original series with regard to a possible radar association, it was certainly necessary to investigate this question further. The initial steps were taken. A replication study was the simplest and least expensive immediate approach. Supplementing it with the independent search of service records added an objective approach eliminating any possible differential in parental responses. These methods having been attempted with inconclusive findings, it is now necessary to look to the prospective, longitudinal, surveillance studies to resolve the issue."

In a study of personnel who had served in the Navy during the Korean War (Silverman, 1979; Robinette and Silverman, 1977), a group of approximately 20,000 persons was selected and classified as having had occupational exposure to RFR on the basis of their titles of Electronics Technician, Fire Control Technician, or Aircraft Electronics Technician; another group of about 21,000 persons was classified as not having had occupational exposure because of their titles of Radioman, Radarman, or Aircraft Technicians Mate. For brevity, the latter group was referred to as the control group, even though these personnel may have had some RFR exposure--presumably much less than the first group. Although comparisons with an unexposed group would have strengthened the study, the two groups selected were presumably similar in terms of non-RFR factors. The study utilized only extant records, covering 1955 to 1976, of mortality and morbidity (both in service and later in Veterans Administration hospitals), and of both granted and disallowed requests for disability compensation.

The report by Robinette and Silverman (1977) provides only mortality results, which show 619 deaths from all causes for the occupationally exposed group versus 579 deaths for the control group, with the difference being not statistically significant. It was noted that these death rates for both groups were lower than those for the comparable age group in the U.S. population at large. Examination of these decedent data in more detail showed a significantly higher death rate from trauma in the exposed group; however, many of the trauma-associated deaths resulted from military aircraft accidents, and a higher proportion of the exposed group had subsequently become flyers. The incidence of deaths associated with arteriosclerotic heart disease was significantly lower in the exposed group. No significant differences were noted between the two groups in terms of total mortality or in terms of mortality from any of about 20 assigned categories of causes of death.

Although the later report by Silverman (1979) does not furnish details regarding morbidity and other health-related aspects, she did state:

> "Differential health risks associated with potential occupational exposure to radar in the Navy more than 20 years ago are not apparent with respect to long-term mortality patterns or hospitalized illness around the period of exposure, two endpoints for which there is virtually complete information for the total study group. Later hospitalization (in Veterans Administration facilities only) and awards for service-connected disability, the two other endpoints examined, provide incomplete information. While some significant differences among the occupational groups classified by level of potential exposure have been found with respect to all the endpoints studied, the differences could not be interpreted as a direct result of microwave exposure."

Peacock et al. (1971) reported that an initial examination of birth certificates filed during the period from July 1969 to November 1970 from Dale and Coffee Counties, Alabama, in which Fort Rucker is located, indicated that the number of clubfoot cases among white babies was much larger than the expected statewide incidence. A more detailed study of this and other congenital anomalies in the six counties surrounding Fort Rucker (Calhoun, Henry, Butler, Jefferson, Dale, and Coffee) showed a higher rate of anomalies among babies born to military personnel than for the state as a whole. However, for non-white populations, only Calhoun County had a significant departure from the expected incidence. No interpretation in terms of causal factors for the excess incidences was given.

Burdeshaw and Schaffer (1977) re-analyzed the Alabama birth record anomaly data for 1968-1972, but instead of using statewide averages as control data, they compared the Coffee and Dale County data with those of each of the other 64 Alabama counties on a score and rank basis. In addition, to acquire more detailed information on hospital characteristics and reporting procedures, they sent questionnaires to 46 Alabama hospitals. They used that information to predict expected values for Lyster General Hospital. They found that the two highest hospital anomaly rates were from Fort Rucker and Maxwell AFB (both military aviation centers) and that 13 of 17 Alabama counties with anomaly rates in the upper quartile were in a contiguous band from southeast to west-northwest Alabama, which indicated the existence of a geographically distributed anomaly problem. However, they also found evidence against the conclusion that there was an unusually high anomaly incidence rate specifically in the Fort Rucker area: overall rates for Coffee and Dale Counties ranked only sixth and eighth among the 67 Alabama counties; at least five other Alabama hospitals reported anomaly incidences that were not significantly lower than for Lyster Hospital; Lyster's overall rate was within predicted limits for hospitals with its characteristics; there was no clustering of residences of mothers with anomalous children in the vicinity of radar sites; carefully controlled surveys from other (non-Alabama) hospitals revealed anomaly incidences consistent with Lyster's; and significant time-clustering of anomalies at Lyster indicated a high reporting rate for one or two particular physicians. In conclusion, they stated that on the basis of the birth record data, it could not be concluded that an unusually large number of infants with congenital anomalies were born to military personnel at Fort Rucker or to other residents in the immediate area.

Siekierzynski (1974) compared the causes of unfitness for work and incidences of lens translucency and of several neurotic disturbances in 507 Polish male radar station workers occupationally exposed to more than 200 $\mu W/cm^2$ with those for a group of 334 men at the same radar stations exposed to less than this value for periods ranging from 2 to 16 years. No correlations were found between the degree of exposure or the duration of employment and any of the criteria of effect. The author states that no appropriate control (unexposed) group was available and that the two groups were highly matched except for exposure intensity.

Kalyada et al. (1974) related narrative clinical evidence indicating that several symptoms were observed in people occupationally exposed to "non-thermal intensities" of RFR at 40 to 200 MHz for periods ranging from 1 to 9 years. The symptoms were described as vegetative dysfunction of the CNS, thermoregulatory pathology, cardiovascular changes, elevation of plasma cholesterol, and gastritis and ulcers. The authors referred to statistically significant changes, but did not present any actual statistical data. They also referred to control subjects, but the incidences of these symptoms in exposed workers were not compared with those in the general population.

Klimkova-Deutschova (1974) surveyed various industrial worker populations, including metal welders, steel factory workers, plastic welders, technicians operating radio or television transmitters, and people working in research institutes and other industries that involve exposure to RFR. Miscellaneous administrative staff members were studied for comparison. Frequencies varied according to the place of exposure, ranging from 1 to 150 MHz, 300 to 800 MHz, or 3 to 30 GHz. The power densities, where specified, ranged from 100 to 3,300 $\mu W/cm^2$. The observations involved 530 people. The findings included electroencephalographic disorders (consisting of synchronized waves of high amplitude and slow rhythm) and biochemical changes (such as elevation of fasting blood glucose, serum beta-lipoprotein, and cholesterol). Changes in brain-wave patterns and in blood sugar, protein, and cholesterol levels were described as more pronounced in the people exposed in the 3-30 GHz range.

Sadcikova (1974) summarized data for two groups of USSR RFR workers; 1,000 people who were subjected to up to a few thousand microwatts/cm^2 and 180 workers who were exposed to up to a few tens of microwatts/cm^2 were compared with a group matched for age and character of work but not exposed to RFR. (Note that although the Soviet occupational standard for exposure to RFR is 10 microwatts/cm^2, Sadcikova was able to locate 1,000 people exposed to up to several hundred times this level.) Of 16 kinds of symptoms reported, the incidences were higher for the higher-power-density group in five cases, higher for the lower-power-density group in nine cases, and essentially equal in two cases. Values of symptoms for the control group were less than those for at least one of the two exposed groups in all 16 cases. Symptoms reported included fatigue, irritability, sleepiness, partial loss of memory, bradycardia, hypertension, hypotension, cardiac pain, and systolic murmur. A table in the report describes 100 cases of "microwave sickness," and the text predicts little chance for recovery unless the patient is removed from the work environment.

Pazderova (1971) reported on the results of a battery of medical evaluations carried out on 58 employees of Czech television transmitter stations. Exposure frequencies were estimated to range from 48.5 to 230 MHz at field intensities equivalent to 0 to 22 $\mu W/cm^2$, with a mean exposure duration of 7.2 years (10.6 hours/workday). Electrocardiograms, heart and lung X-rays, erythrocyte sedimentation rates, urinalyses, and liver function tests were conducted as well as hematologic, serologic, ophthalmologic, neurologic, gynecologic, psychiatric, and psychologic examinations. The only statistically significant finding was that the mean plasma protein levels were higher than "normal" values taken from the literature, a finding that even the author finds unexplainable. The appropriateness of the use of literature control values is highly questionable, and the author notes the desirability of a control group matched for "age, way of life and educational background."

The U.S., Polish, and Czechoslovakian studies offer no evidence of detrimental effects associated with exposure of the general population to RFR. Consistent with the voluminous, earlier Soviet literature, the Soviet studies offer findings that occupational exposure to RFR at average power densities less than 10 mW/cm^2 does result in various symptoms, particularly those associated with CNS disorders. Because the USSR symptomatology has never been reported in Western studies and because there are marked differences between Soviet and Western publications in the procedures used for reporting data, any prediction of possible RFR hazards based on the USSR epidemiological studies would require acceptance of these Soviet findings at face value.

Overall, the epidemiological evidence does not offer any reliable evidence that the ordinary level of exposure of humans within the rectenna area of SPS (0.01 to 1.0 mW/cm^2) would cause any harm. The major exception is that persons required to climb above the rectenna system might be exposed to as much as 22 mW/cm^2, and for this level of exposure there are no human data.

6.2 GENETIC AND CYTOGENETIC EFFECTS AND CANCER INDUCTION

Although a number of papers published over the last 30 years have claimed that microwave irradiation at various frequencies can produce mutations, chromosome aberrations, and cancer, a careful review of the various papers indicates that all of the reported effects are probably due to temperature rise, faulty experimental procedure, or other incidental causes.

Direct mutation studies have been conducted in bacteria, fruit flies, and mice. The study in bacteria (Blackman, 1976) involved exposure of *E. coli* WWU to 1.70 or 2.45 GHz at 2 to 50 mW/cm^2 for 3 to 4 hours. The results were negative--there was no increase in mutations over controls. Two separate studies in *Drosophila* (Mickey, 1974; Dardalhon, 1977) also had negative results. A third study (Pay and Beyer, 1972) found no evidence of microwave-induced mutation in *Drosophila* and also found no consistent evidence of an effect of microwaves on reproductive capacity of *Drosophila*. Two studies of mutagenesis in mice, using a dominant lethal assay, gave conflicting results. In the first study (Varma et al., 1975a, 1975b), male mice were exposed to microwave radiation (1.7 GHz, 50 mW/cm^2, 30 min, or 10 mW/cm^2, 80 min; and 2.45 GHz, 100 mW/cm^2, 10 min, or 50 mW/cm^2 for 3 X 10 min in one day or 4 X 10 min in 2 weeks) and then bred to unexposed females once a week for the next 7 to 8 weeks. The females were killed on the 13th day of gestation, and the uteri were removed and examined for numbers of implants and early embryonic deaths. The authors concluded that the 1.7-GHz radiation was mutagenic at both power-density levels, but that the 2.45-GHz radiation was mutagenic only for the 100 mW/cm^2 power-density level. In the second study (Berman and Carter, 1978), mice were exposed to microwaves (425 MHz and 2.45 GHz, 5 to 28 mW/cm^2) in repeated daily exposures for periods of up to 3 months. The authors found no evidence of either dominant lethal mutations or reduced fertility. Review of the first study shows that there was a discrepancy in the rate of occurrence of spontaneous mutations in control mice for the two power-density levels

(1% vs 5%) and an error in the statistical computations. Taken as a whole, the study indicates that there might have been a marginal increase in early fetal deaths (dominant lethals) at 1.7 GHz; however, because of the discrepancy in control values at the two frequencies, the result can be considered unproven. Considering both studies together, it appears likely that mutagenic effects in mice, if they exist at all, are the result of temperature rise in the testes rather than showing any intrinsic mutagenic potential of the microwaves.

Cytogenetic studies have included observation of chromosome aberrations in a variety of cell types and sister chromatid exchange in Chinese hamster ovary cells. In the study of sister chromatid exchange (Livingston et al., 1977), exposure of cells in vitro to 2.45-GHz microwaves did cause a rise in number of exchanges, but the effect was clearly the result of a rise in the temperature of the medium. Two studies reported observation of chromosome aberrations in cells following microwave exposure of the cells in vitro. In the first study (Chen et al., 1974), Chinese hamster cells and human amnion cells were exposed to 2.45-GHz microwaves at 200 to 500 mW/cm^2 for 1.5 to 20 minutes. There was no significant difference in the incidence of chromosome aberrations between control cultures and cultures exposed at any dose level for either cell type. In the second study (Stodolnik-Baranska, 1974b), human lymphocyte cultures were exposed to 2.95-GHz microwaves at 20 or 7 mW/cm^2. Exposure at 20 mW/cm^2 caused a rise in temperature of the culture medium and an increase in the number of chromosome aberrations, but neither of these effects occurred on exposure at 7 mW/cm^2.

Chromosome aberrations have also been reported in cells of garlic root-tips and in lung and bone marrow cells and spermatogonia of Chinese hamsters exposed to microwaves in vivo (Heller, 1959; Mickey et al., 1975). The dosimetry is described very poorly in both reports, and the experimental procedures are described very poorly in the second report; hence evaluation of the work is rather difficult. However, the studies involved exposures in the region of 10 to 30 GHz at power-density levels ranging up to 600 mW/cm^2 for long periods of time. Again, it appears doubtful that microwaves actually caused any chromosomal aberrations. Considering all of the studies together, the results indicate that, where the reports are reliable, the cytogenetic effects of microwave radiation are solely the result of heat produced by the irradiation and do not indicate any intrinsic mutagenic potential of microwaves.

Although mutagenic effects of environmental agents are considered potentially deleterious, the principal reason for concern about mutagenesis at the present time is that it may indicate carcinogenic potential (Ames et al., 1979). A review of the literature reveals only three papers indicating the possibility of cancer induction by microwave irradiation. The first paper (Zaret, 1976) stated that the incidence of cancer in the human population of the Karelia district of Finland had increased following installation of an extensive radar warning system in an adjacent region of the USSR. Since the statement was not supported by any data on cancer incidence, it can be disregarded. The second paper (Prausnitz and Susskind, 1962) reported a somewhat higher incidence of leukosis in mice chronically exposed to microwaves (9.27 GHz, 100 mW/cm^2, 4.5 minutes/day, 59 weeks), which was described as "cancer."

In reviewing the reported finding, the following points have been noted:

- Tissues from a number of animals dying during the study were lost because of autolysis. Hence the finding of leucosis is based on incomplete groups.
- Leucosis means an abnormally high leukocyte count in the circulating blood, and can arise from a variety of causes, including--possibly--action of microwaves on the immunological or endocrine systems.
- Some of the mice in the study were sacrificed, and sections of spleen were examined for presence of leukemia. There was no difference in leukemia incidence between experimental and control animals.

- The authors noted that the survival of the microwave-exposed animals was somewhat longer than that of the controls, and speculated on whether a brief daily exposure to microwaves might actually be beneficial to animals.

The third paper (Dwyer, 1978) is a review of the potentially carcinogenic properties of microwave irradiation prepared for the National Institute of Occupational Safety and Health under contract with the Franklin Institute. The review covers most of the papers reviewed in this section, but contains no critical analysis of power density levels, sources of data, reliability of conclusions, or validity of experimental procedures; hence no inference about potential carcinogenicity of microwaves can be drawn from it.

In conclusion, there is no reliabble or systematic evidence that microwave irradiation can induce any type of mutation in living systems other than--possibly--by induction of heat in the tissue under examination. The mechanism by which temperature rise could induce apparent mutagenic effects is not understood. Possibly an increase in temperature accelerates the rate of spontaneous mutagenic processes in the tissue. There is no evidence that microwave irradiation induces cancer. On the whole the specter of mutation or cancer from microwave irradiation can probably be laid to rest as nonexistent.

Since there appear to be no intrinsic mutagenic or carcinogenic effects of microwaves, the problem of such effects can be dismissed for the ordinary level of exposure of humans within the rectenna area of SPS (0.01 to 1.0 mW/cm^2). For persons who might occasionally be exposed above the rectenna to the full beam power (22 mW/cm^2), it is hypothetically possible that heat-induced mutations or cytogenetic effects might occur. However, only one out of seven biological studies reported mutations, and all of the cytogenetic studies involved isolated cells that lacked any capacity for control of temperature rise, such as would be found in mammalian tissues *in vivo*. Hence, induction of mutations or cytogenetic effects in humans by the full beam power of SPS must be considered highly improbable.

6.3 STUDIES ON TERATOGENESIS AND DEVELOPMENTAL ABNORMALITIES

In the narrowest sense of the word, teratogenesis refers to the production of anatomical aberrations in a developing fetus. The term is most often applied to development of mammalian fetuses, but studies of development of eggs of birds and the pupae of the darkling beetle, *Tenebrio molitor*, have also been performed with microwave radiation. Although the term usually refers to anatomical anomalies, studies have also included observation of fetal death and/or resorption and of physiological and cellular abnormalities in the offspring observed postpartum.

Two critical remarks are pertinent to studies of teratogenesis in general. First, the ordinary mechanism by which terata are usually produced involves alteration (often temporary) in the rate of growth of a particular tissue under development. Development of the entire fetus is a complex process requiring that individual tissues develop within a preset time frame, and interruption of this timing will result in abnormalities because a particular tissue or organ fails to complete development on schedule. Because of this, production of abnormalities is highly dependent on time in the gestation sequence when the agent is applied and on the species of animal under study. The second remark is that there is a difference in experimental circumstances in studying development of birds' eggs or insect pupae, as contrasted with the study of mammalian teratogenesis. In the former instance, the experimental material is exposed to the whole environment without any protection; hence the studies must include rigorous control of all environmental parameters, including temperature. In the latter instance, the developing fetus is isolated from the environment to some extent by the dam; however, influences of the noxious agent on the dam must be considered as a potential indirect source of teratogenic effect.

A number of studies have been conducted on the induction of teratogenesis in pupae of *Tenebrio*. The two most recently published studies (Green et al., 1979; Pickard and Olsen, 1979) will be discussed here. These studies reviewed previously published work and commented on the variability and inconsistency of results. Green and co-workers showed that teratogenic effect was partly dependent on the humidity of the environmental chamber, which affected the rate of loss of water from the pupae during irradiation. They observed that there was a slight increase in incidence of terata with increasing applied microwave power (2-hour constant exposure) up to 40 mW of applied power. At 80 mW there was a decrease in teratogenic frequency, and at 160 and 320 mW there was a further increase in teratogenic frequency, accompanied by an increase in death of pupae before completion of development. The authors attributed the apparent "power window" at 80 mW to an antagonism between nonthermal teratogenic effects and protective effects caused by the rise in temperature. The second study showed that the incidence of teratogenesis in the pupae depended on a number of factors, including the source of the larvae, the diet on which they were raised, and the conditions under which the pupae were maintained during development.

Taken together, the studies indicate that teratogenesis in Tenebrio pupae depends on such a wide variety of environmental influences that the use of such a model for studying microwave teratogenesis is questionable. The existence of the "power window" at 80 mW is quite interesting, but the effect can be explained by a number of possible causes other than that offered by the authors. In the first place, the assertion of nonthermal effects below 80 mW is unproven, since the authors show (Figure 5, p. 169 in Green et al., 1979) that 40 mW of power produces a rise in temperature. One possible explanation is that at 80 mW under the conditions of the experiment, the irradiation induces a water loss from the pupae to a level that renders the pupae relatively resistant to teratogenesis.

Fisher et al. (1979) studied the development of chicken embryos during a 4- or 5-day continuous exposure to microwaves (2.45 GHz, 1.4 and 6.2 mW/cm^2). The eggs were exposed to a gradient of incubator temperatures from 32 to 36°C, and the stage of development of the embryos was observed at the end of the exposure. In the control eggs, the rate of development was dependent on the temperature; however, this was not so for the microwave-exposed eggs. The authors observed no difference in incidence of sterility or premature deaths between the two groups. From the description of the method it is difficult to determine whether the temperature of the microwave-exposed eggs were actually measured or whether possible differences in temperatures of microwave-exposed eggs might have existed because of inhomogeneity of the microwave field. Finally, the significance of the finding in relation to possible human hazard is unclear, since the embryos were not carried to hatching.

In Japanese quail embryos exposed to continuous microwave radiation (2.45-GHz, 5 mW/cm^2) during the first 12 days of development, there were no gross deformities at hatching (McRee and Hamrick, 1977). The investigators did find a slight increase in blood hemoglobin and a slight decrease in monocyte count in the exposed birds, as compared with controls. A further study (Hamrick et al., 1977) showed that exposed male birds were slightly smaller than control birds at 4 and 5 weeks of age. The significance of these findings for human health is questionable.

Teratogenic effects of microwaves have been reported for a number of studies in rats and mice. The subject was reviewed recently by O'Connor (1980), who observed that, because of the high power density levels employed, the probability of killing the dam was somewhat larger than the probability of producing a teratogenic effect. The only major study in which a significant number of terata were produced without significant mortality of the dams was that of Rugh et al. (1975). These researchers exposed pregnant CF-1 mice on the eighth day of gestation to 2.45-GHz radiation at a power density level of about 120 mW/cm^2 for two to five minutes. Mice were sacrificed on the 18th day of gestation, and the fetuses were examined for gross abnormalities and evidence of resorption. The authors described several visible abnormalities, but reported only on the incidence of exencephaly--an abnormality in which the skull fails to close, leaving the brain exposed. There appear to have been no controls; however, exencephaly is a comparatively rare anomaly, so the absence of control incidence is, in this study, probably inconsequential.

The authors claimed that microwave radiation did not produce a linear relationship between exencephaly and absorbed dose, and that there was no evidence of a threshold effect. To test these statements, we extracted the data on incidence of exencephaly (Figure 8, page 232 of the paper) and re-analyzed them. The doses and incidences of exencephaly were determined for each point in the figure and, from the litters showing exencephaly, the numbers of affected pups and litter sizes were calculated. Litter size could be determined only from the litters showing partial response, but the mean value of 56 such litters was 10 pups per litter. This value was used in all subsequent calculations.

The microwave doses in the experiment were spread out more or less on a continuum from 3 to 8 Cal./gram. To facilitate analysis, we grouped the litters by dose intervals of 0.5 Cal./gram, and the mean dose of the interval. Some inaccuracy may have arisen in the count of the number of zero-responders, because points for these litters were plotted quite densely along the X-axis of the figure. Where the plot was represented by a solid line, we assumed that there was one litter for each 0.045 Cal./gram dose interval. It is doubtful that the uncertainty of litter count would significantly affect the conclusions of our analysis.

The results of this analysis are presented in Table 6-1 and Figure 6-1. When the results are considered in terms of fraction of litters showing exencephaly, the incidence increases with increasing dose to a level of about 50% of the litters, after which there is no systematic increase. When the results are considered in terms of the fraction of the total pups affected, the incidence rises linearly with increasing dose over the entire range. From both viewpoints of analysis, there is an apparent threshold at slightly less than 4 Cal./gram. From a linear regression of the points for fraction of

Table 6-1

INCIDENCE OF EXENCEPHALIES IN MICE EXPOSED IN UTERO TO MICROWAVE RADIATION ON THE EIGHTH DAY OF GESTATION

Dose Range Cal./g	Mean Dose* Cal./g	No. of Litters	No. of Pups	Litters with Exencephaly		Pups with Exencephaly	
				No.	Fraction	No.	Fraction
3.0-3.45	3.32	6	60	0	0	0	0
3.5-3.95	3.72	10	100	0	0	0	0
4.0-4.45	4.24	13	130	2	0.154	2	0.0154
4.5-4.95	4.72	15	150	4	0.267	6	0.0400
5.0-5.45	5.27	15	150	5	0.333	7	0.0467
5.5-5.95	5.74	22	220	11	0.500	17	0.0773
6.0-6.45	6.27	20	200	9	0.450	17	0.0850
6.5-6.95	6.75	22	220	12	0.546	17	0.0773
7.0-7.45	7.18	16	160	8	0.500	21	0.1313
7.5-7.95	7.61	11	110	5	0.454	12	0.1091

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* Extracted from Rugh et al. (1975). Standard deviation range: 0.10-0.16.

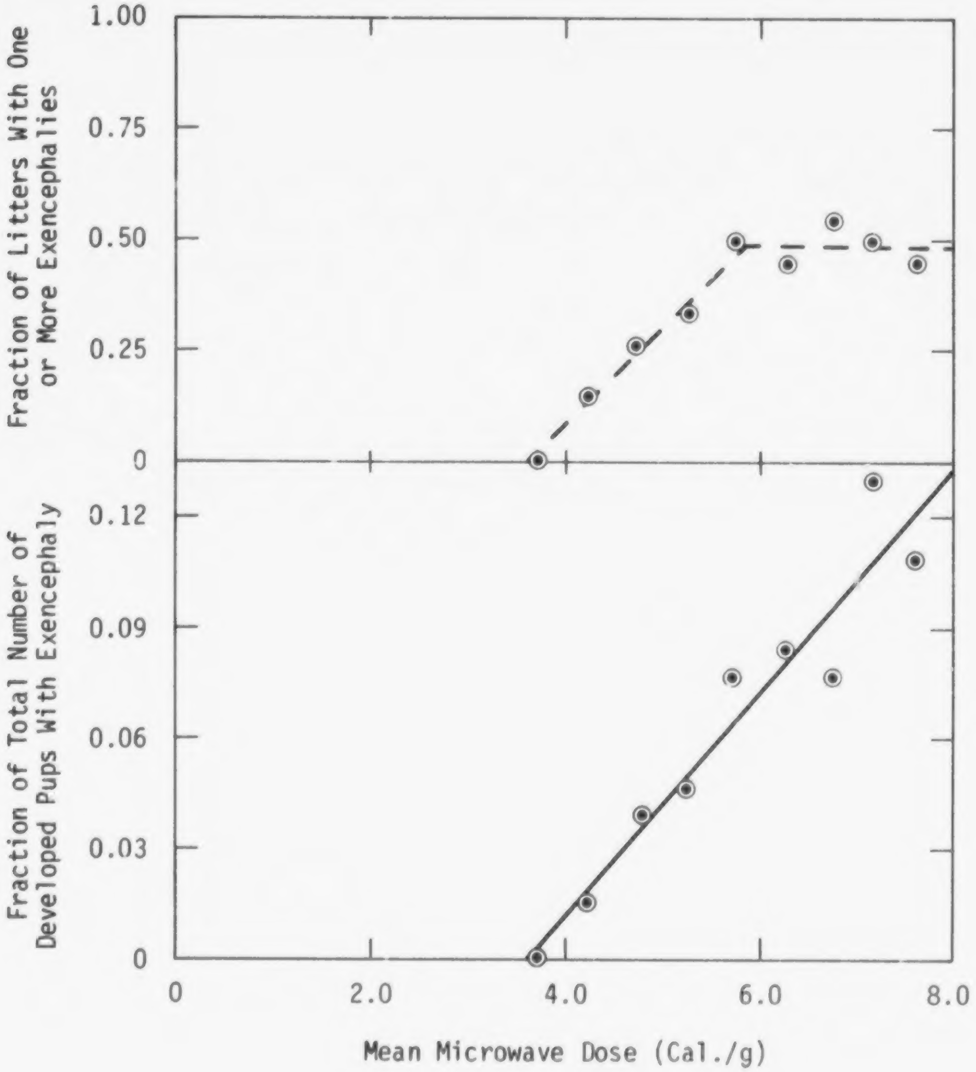

FIGURE 6-1 PRODUCTION OF FETAL EXENCEPHALIES IN MICE BY EXPOSURE TO MICROWAVE RADIATION (adapted from Rugh et al., 1975, Figure 8, page 232)

affected pups on dose, the apparent threshold dose was calculated to be 3.6 Cal./gram.

The incidence of resorption sites in the uteri of microwave-exposed mice (Figure 8, page 231 of the paper) was also analyzed by the procedures described above. The results of the analysis are presented in Table 6-2 and Figure 6-2. Whether the incidence of resorptions is considered in terms of fraction of the number of litters affected or fraction of the total number of implantations affected, the result is the same: the incidence rises linearly with increasing microwave dose. Again, there is a suggestion of a dose threshold, calculated to be between 1.5 and 3.5 Cal./gram.

Another extensive study of teratogenesis in mice was conducted by Berman et al. (1978), who exposed mice to 2.45-GHz microwaves at power-density levels of 3.4 to 28 mW/cm^2 for 100 minutes per day during most of the period of gestation. The study found 27 anomalies among 3,362 live fetuses exposed to microwaves, as compared to 12 among 3,528 sham-irradiated controls. Overall, this would indicate a marginal teratogenic effect of the microwaves. Because of the small incidence of each individual anomaly and the absence of a consistent dose-response pattern, it is impossible to demonstrate conclusively that the anomalies result from the microwave exposure.

Several studies have been conducted or are in progress (Reiter, 1976; Rosenstein, 1976; Kaplan et al., 1978) to determine effects of microwave exposure _in utero_ in the power-density range of 0.1 to 10 mW/cm^2 on the neurological and behavioral development of animals following parturition. Full reports have not been available for review, but abstracts or summaries have indicated negative results.

In summary, teratogenic effects of microwave radiation appear to occur reliably only at relatively high power-density levels, probably greater than 28 mW/cm^2. In relation to the problem of SPSS, it appears

Table 6-2

INCIDENCE OF RESORPTION SITES IN UTERI OF PREGNANT MICE EXPOSED TO MICROWAVES ON THE EIGHTH DAY OF PREGNANCY

Dose Range Cal./g	Mean Dose* Cal./g	No. of Litters	No. of Pups	Litters with Resorptions		Pups Resorbed	
				No.	Fraction	No.	Fraction
3.0-3.45	3.27	1	10	0	0	0	0
3.5-3.95	3.72	16	160	5	0.312	7	0.0438
4.0-4.45	4.23	17	170	6	0.353	10	0.0588
4.5-4.95	4.76	23	230	12	0.522	32	0.1391
5.0-5.45	5.30	29	290	20	0.690	43	0.1483
5.5-5.95	5.70	32	320	24	0.750	91	0.2844
6.0-6.45	6.23	20	200	15	0.750	64	0.3200
6.5-6.95	6.70	25	250	23	0.920	74	0.2960
7.0-7.45	7.20	16	160	15	0.938	65	0.4062
7.5-7.95	7.59	8	80	5	0.625	25	0.3125

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* Extracted from Rugh et al. (1975). Standard deviation range: 0.08-0.15.

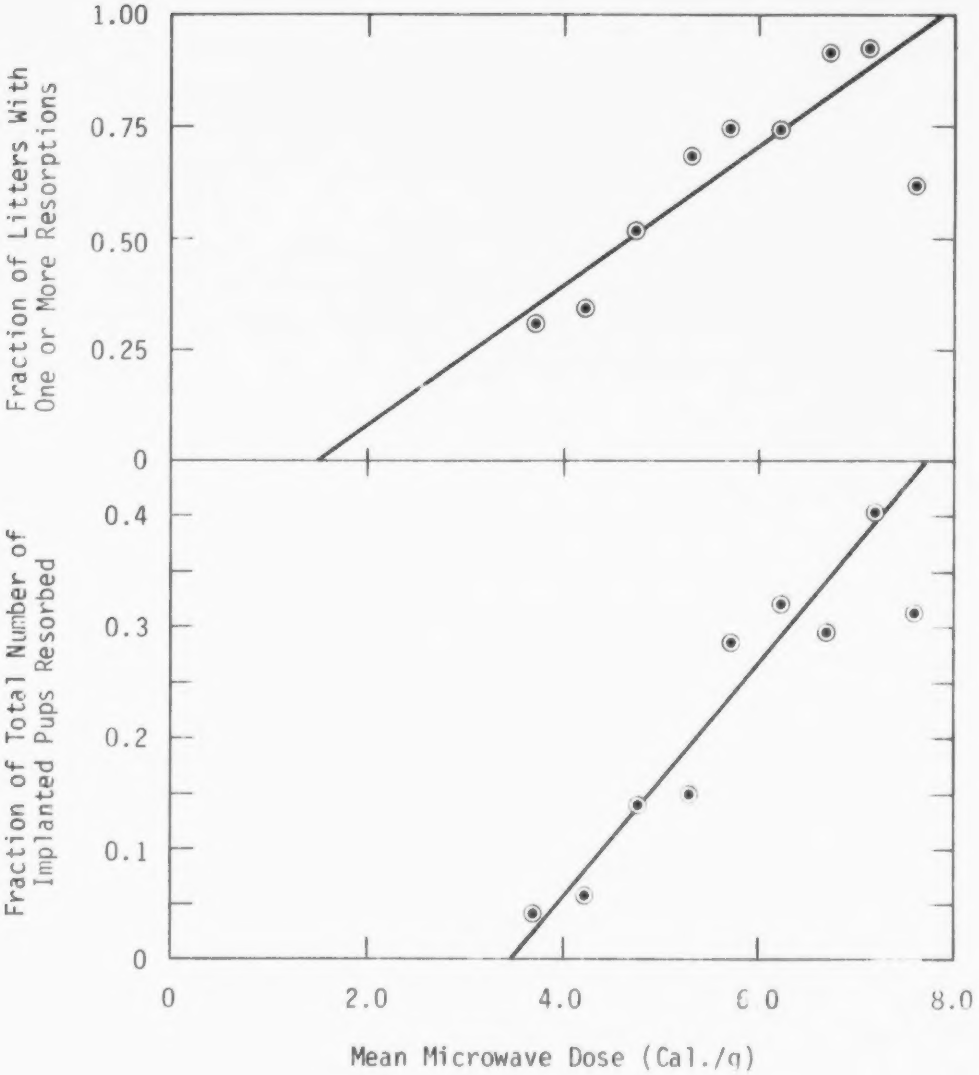

FIGURE 6-2 PRODUCTION OF FETAL RESORPTIONS IN MICE BY EXPOSURE TO MICROWAVE RADIATION (adapted from Rugh et al., 1975, Figure 7, p. 231)

very likely that neither the ordinary level of exposure of humans (0.01 to 1.0 mW/cm^2) nor the maximum level (22 mW/cm^2) would cause any teratogenic effect in humans.

6.4 OCULAR EFFECTS

6.4.1 Animals

During the past 30 years, various investigations have been conducted on the effects of RFR exposure on the eyes of live experimental animals. Many of the results indicate that intraocular temperature increases of about 5°C or more are necessary for eye damage (Guy et al., 1974; Williams et al., 1955; Daily et al., 1952; Richardson et al., 1948). Also, lens opacifications caused by RFR exposure alone were not produced at the same average power density when the eye was cooled (Kramar et al., 1975; Baillie et al., 1969a, 1969b).

Many of the results of RFR exposure indicate the reciprocity (inverse relationship) between average power density and exposure duration for cataract formation and the existence of a threshold average power density of about 150 mW/cm^2 for single or multiple exposures for tens of minutes or more (Carpenter, 1977; Ferri and Foti, 1977; Guy et al., 1969; Williams et al., 1955). As a representative example (Guy et al., 1974), for average power densities decreasing from about 500 to 200 mW/cm^2, the exposure duration needed for causing eye damage in the rabbit increased from 1-2 minutes to about 20 minutes. Also, cataracts were not produced at 100 mW/cm^2 for exposure durations of up to at least 100 minutes (exposures for longer periods were not done in this investigation). Thus, curves of average power density versus exposure duration show that the average power density for cataractogenesis asymptotically approaches a value of about 150 mW/cm^2 (the aforementioned threshold). Cataractogenesis thresholds of comparable magnitude are evident from the experimental results of others (Carpenter, 1977; Birenbaum et al., 1969; Williams et al., 1955). Carpenter (1979), in reviewing RFR cataractogenesis from a clinical viewpoint, presents a detailed description of the post-irradiation progression of eye changes, based on experimental results with animals.

Kramar et al. (1975) measured intraocular temperatures *in vivo* at 200 mW/cm^2 over a period of 40 minutes by quickly inserting a thermocouple probe during brief shut-off of the RFR at the end of successive five-minute exposure periods. They found that the temperature of the vitreous humor rose from about 37°C to about 42°C during the first 10 minutes of exposure and remained at 42°C for the rest of the exposure period. Equilibrium between the rates of energy absorption and heat removal in that region of the eye is believed to be the determining factor in attaining the 42°C plateau temperature. In the orbit, which is cooled by blood flow to a greater extent than the vitreous humor, the corresponding plateau temperature was less than 40°C.

The effects of temperature increases produced by non-RFR means on isolated (*in vitro*) rat lenses were studied recently by Stewart-DeHaan et al. (1979). Lenses incubated in tissue culture medium M 199 (containing 10% fetal calf serum) at the normal physiological temperature of 37°C maintained their clarity for up to two weeks. When ten times the normal concentration of serum glucose was included in the medium, opacities and associated globular degeneration of the lens cells developed in one day. Similar effects were observed when lenses were warmed to 39 or 41°C for one hour and then incubated at 37°C for 24 hours. Lenses heated to 60 or 75°C for one hour did not become opaque. The authors surmise that the lenses became histologically "fixed." Radioactive tracer studies suggest that membrane changes may be involved in temperature-induced cataract formation.

A number of investigators (Ferri and Foti, 1977; Reider et al., 1971; Richardson et al. 1951) compared the ocular effects of pulsed and CW RFR at equivalent average power densities. In representative investigations, the average power densities were greater than 100 mW/cm^2 and the exposures were for about 1 hour/day for several weeks. No significant differences between the effects of pulsed and CW RFR were found.

The existence of a cataractogenesis threshold of about 150 mW/cm^2 is regarded as evidence that single or multiple exposure for indefinitely long durations at average power densities well below the threshold would not cause eye damage to humans or any other species.

This conclusion is supported by preliminary results of an investigation by Chou et al. (1978). They exposed one group of six rabbits to 2.45 GHz CW at 1.5 mW/cm^2 for two hours per day over several months and another group to pulsed RFR at the same frequency and average power density (10-μs pulses at a repetition rate of 100 pulses/s, comprising a duty cycle of 0.001 and a pulse power density of 1.5 W/cm^2); a third group was sham-irradiated. Periodic eye examinations for cataract formation yielded no statistically significant differences among the three groups. (This investigation also included comparisons of body weights, EEG and evoked potentials, hematology, and blood serum chemistry; no significant differences were found among the groups for these aspects either.)

6.4.2 Humans

Some cases of ocular damage in humans ascribed to occupational exposure to RFR were reported during the 25 years following the end of World War II (Zaret, 1969; Shimkovich and Shilyaev, 1959; Hirsh and Parker, 1952). Although the exposure histories of these individuals could not be ascertained with any degree of certitude, it is likely that their actual or incipient vision impairment was due to exposure to average power densities well above the threshold.

More recently, the occurrence of cataracts in two editors with the New York Times was ascribed, in newspaper accounts during 1977 and 1978, to their exposure to supposed RFR from the cathode-ray tubes in video-display terminals used by them (Justesen, 1979). (Cases of RFR-induced birth defects and abortions were also linked, in other newspaper stories, to exposure to video terminals.) The New York Times arranged for measurement surveys of the terminals in question. These surveys yielded negative results; the only measurable radiations emitted by the terminals were well above the RFR spectrum (primarily in the visible range). Independent surveys of the same terminals by personnel from the National Institute for Occupational Safety and Health (1977, cited in Justesen, 1979) confirmed these findings.

Several retrospective epidemiological studies were performed, notably by Zaret et al. (1961), Cleary et al. (1965), and Appleton (1973), to ascertain whether chronic exposure to RFR could cause cataracts.

Zaret et al. (1961) examined a group of 475 persons for eye defects, who were believed to have been exposed to RFR at 11 military and non-military establishments, and a group of 359 persons as controls. The investigators found a slight but statistically significant difference in defect scores between the two groups, but expressed some doubt regarding the full validity of the scoring method used.

Cleary et al. (1965) examined Veterans Administration Hospital records of 2,946 Army and Air Force veterans of World War II and the Korean War who had been treated for cataracts. A control sample of 2,164 veterans was selected. Based on military occupational specialties, they classified each individual as a radar workers, or as nonradar workers, or one whose specialty could not be discerned. In the radar group, they found 19 individuals with cataracts and 2,625 individuals without cataracts, whereas in the nonradar group, there were 21 individuals with cataracts and 9,935 individuals without cataracts. (The remaining 510 individuals were in the unspecified occupational category.) These differences between the radar and nonradar groups are not statistically significant. Cleary and Pasternack (1966) statistically analyzed the records of 736 microwave workers and 559 control persons for minor lens changes, using a scoring range from 0 to 3. They reported that the defect scores increased with age for persons in both groups, but that the average score for the microwave group was significantly higher than for the control group. They suggested that this finding is an indication that exposure to RFR may have an aging effect on the lens. However, no cataracts or decrease of visual acuity were found.

In the Appleton (1973) study, which covered a period of five years, military personnel identified as having been occupationally exposed to RFR from radar and communications systems were matched as closely as possible in age and sex with other military personnel on the same bases who had not been occupationally exposed. Several ophthalmologists independently examined exposed and control personnel (without knowledge as to the group to which each individual belonged) for opacities, vacuoles, and posterior subcapsular iridescence, taken as diagnostic precursors of cataracts. Because of the complexity of the eye and the unavoidable judgmental aspects in the diagnosis of each examining ophthalmologist, each precursor was scored as either present or absent in each individual, and the binary data thus obtained were used for statistical analyses by age group and numbers of persons per age group. The results indicated that more people in older age groups exhibited these precursors, but the pooled data from several Army installations showed no statistically significant differences between exposed and control groups. The presence or absence of the three diagnostic precursors is only a crude measure of actual or possible incipient eye damage, useful primarily for statistical purposes. Nevertheless, the findings of no statistical differences between exposed and control groups are probably valid.

The principal investigator recognized the judgmental aspects by citing the results of two of the ophthalmologists who had examined the personnel at Tyndall AFB. These ophthalmologists scored the presence of opacities in 96% of the personnel in the exposed group and 93% in the control group. However, they also concluded that "no optically significant opacities were found in either group," which illustrated the point emphasized by the principal investigator that the presence of any of the precursors did not necessarily mean that significant vision impairment had occurred.

As with other retrospective epidemiological studies, the extent of the exposure histories (frequencies, intensities, durations, etc.) in either the exposed or the control groups used in these three studies is difficult to determine accurately. However, it is quite likely that the exposed groups did receive more RFR exposure than the control groups.

The results support the conclusion from animal studies that chronic exposure to RFR at average power densities well below the threshold is not likely to produce cataracts. The threshold power density indicated from the animal studies is of the order of 100 mW/cm^2 or more. Within the rectenna area of SPS, neither the exposure (0.01 to 1.0 mW/cm^2) nor the maximum exposure (23 mW/cm^2) has any realistic probability of inducing cataracts in workers in the area.

6.5 NERVOUS SYSTEM STUDIES

6.5.1 RFR Hearing Effect

Humans in the vicinity of some types of pulsed radar systems have perceived individual pulses of RFR as audible clicks (without the use of any electronic receptors). This phenomenon, first investigated by Frey (1961), has attracted much interest--especially in the U.S.--because it has often been cited as evidence that nonthermal effects can occur and because an initial hypothesis was that one of the possible mechanisms for perception is direct stimulation of the CNS by RFR. Various theoretical and experimental studies, the latter with both human volunteers and laboratory animals, have been conducted to determine the conditions under which pulsed RFR can be heard and to investigate the interaction mechanisms involved. Many of the results support the hypothesis that a pulse of RFR having the requisite pulse power density and duration can produce a transient thermal gradient large enough to generate an elastic shock wave at some boundary between regions of dissimilar dielectric properties in the head, and that this shock wave is transmitted to the middle ear, where it is perceived as a click. Persons with impaired hearing are unable to hear such clicks, and experimental animals in which the cochlea (the inner ear) has been destroyed do not exhibit brainstem-evoked responses. Therefore, this phenomenon is unlikely to be attributable to direct RFR stimulation of the brain. Also, because individual pulses can be perceived, citations of low average power densities based on calculations for two or more widely spaced pulses are not particularly meaningful. Representative investigations of this phenomenon are summarized below.

White (1963) reported that acoustic transients can be generated in various metals and plastics, in a piezoelectric crystal, and in liquids such as water by transient surface-heating with pulses of RFR (or from an electron beam). Such transients were detectable in a barium titanate crystal at calculated surface temperature rises of the order of only 0.001°C. Foster and Finch (1974), using 2.45 GHz RFR, confirmed White's findings that such transients can be produced in water, and they measured the audiofrequency pressures generated by several combinations of pulse power density and pulse duration. The results indicated that the peak pressures would be sufficient to induce human perception of such RF pulses as auditory clicks. They also showed that the effect vanished when the water was cooled to 4°C, where its thermal expansion coefficient is very small. These results support the thermoelastic hypothesis.

Lin (1977a, 1977b) has reported on detailed theoretical studies of the RFR auditory effect. He assumed that the auditory sensation results from acoustic waves generated in the tissues of the head by rapid thermal expansion of the tissues upon microwave absorption, in consonance with the investigations cited above. His results indicate that the audio frequencies produced are independent of the frequency of the RFR, but are dependent on head size. The predicted fundamental frequency is 13 kHz for an adult human and 18 kHz for an infant.

Chou, Guy, and Galambos (1977) studied cochlear microphonics (CM) produced by pulsed 918- and 2450-MHz RFR in guinea pigs and cats. They found that the CM frequencies correlated well with the longest dimension of the brain cavities of the two species, but poorly with other brain cavity dimensions. Extrapolation of the results indicates that CM frequencies in humans should be between 7 and 10 kHz, in reasonable agreement with Lin's theoretical calculations.

Chou and Guy (1979b) used 918-MHz RFR to investigate the thresholds for brainstem-evoked responses in guinea pigs. They found that for pulse durations of 10, 20, and 30 μs, the threshold incident energy density was approximately constant (1.56-1.87 $\mu J/cm^2$ per pulse), corresponding to incident pulse power densities of 156, 78, and 62.4 mW/cm^2, respectively. However, for pulse durations longer than 70 μs, the incident pulse power density necessary for obtaining responses was approximately constant (about 90 mW/cm^2), representing corresponding increases of incident energy density per pulse with pulse duration. Chou and Guy indicated that their experimental results agree well with the predictions of the thermal expansion theory.

Chou and Galambos (1977) investigated the effects of external-ear blocking, middle-ear damping, and middle-ear destruction on brainstem-evoked responses to both acoustic and RFR stimuli. They found that the animal was capable of responding to pulsed RFR only if its cochlea was intact.

Lebovitz and Seaman (1977) reported on single auditory unit (eighth-nerve-related) responses to pulsed 915-MHz RFR and acoustic clicks. They found that the response of a typical single auditory unit was very similar to the response of the unit to acoustic click stimuli, differing primarily only in amplitude.

Cain and Rissman (1976, 1978) used 3.0-GHz RFR to study the auditory effect in two cats, two chinchillas, one beagle, and eight human volunteers. For the animals, surface or brainstem-implanted electrodes were used to measure the responses to RFR pulses and the responses evoked by audio clicks from a speaker. They found that the threshold energy density for RFR responses ranged from 8.7 to 14 $\mu J/cm^2$ per pulse for the cats, from 7.5 to 20 $\mu J/cm^2$ for the chinchillas, and averaged 5.0 $\mu J/cm^2$ for the beagle. For a pulse width of 10 μs, these values correspond to pulse power densities of 1.3 W/cm^2 for both cats, 1 and 2 W/cm^2 for the two chinchillas, and 300 mW/cm^2 for the beagle. The eight humans were given standard audiograms. Because such audiograms do not test hearing above 8 kHz, binaural hearing thresholds were also determined for seven of the subjects for frequencies in the range from 1 to 20 kHz. Five of the subjects could detect 15-μs pulses as clicks; the other three required a pulse duration of 20 μs for perception. No correlation between the results and the audiograms was apparent; however, there was a strong correlation between RFR perception and hearing ability above 8 kHz as determined from the binaural thresholds. The average threshold energy density for the humans was 10.6 $\mu J/cm^2$ per pulse. For 15-μs pulses, this value corresponds to a pulse power density of about 700 mW/cm^2; however, three of the subjects were able to perceive 15-μs pulses at a pulse power density of 300 mW/cm^2.

Tyazhelov et al. (1979) reported some peculiarities regarding auditory perception of pulsed 800-MHz RFR in humans. The pulse widths used ranged from 5 to 150 μs at pulse repetition frequencies (prfs) of 50 to 2,000 (the latter for short pulse durations). Each subject could be presented with sinusoidal audiofrequency (AF) sound waves alternately or concurrently with the pulsed RFR and could adjust the amplitude, frequency, and phase of the AF signal to match the timbre and loudness of the perceived RFR. Using AF signals, the high frequency auditory limit (HFAL) of each subject was tested first. Three of the subjects had a HFAL below 10 kHz and could not perceive RFR pulses of short duration (10 to 30 μs), whereas only one of the 15 subjects having a HFAL above 10 kHz could not perceive such pulses. These results are consonant with those of Cain and Rissman. Among the peculiarities noted by Tyazhelov et al. was the biphasic dependence of RFR perception thresholds on pulse width. They also reported that subjects could detect beat frequencies when concurrently presented with 8-kHz AF sound and RFR having prfs above or below 8 kHz, and that when the prf was the same as the AF, the subject could cancel perception by adjusting the phase of the AF. Their conclusion is that the thermoelastic model is inadequate to explain these observations.

Frey and Coren (1979), using dynamic time-averaged interferometric holography, endeavored to detect tissue movement in successive layers of heads of animals exposed to pulsed RFR; for comparison, they used holograms obtained for the same animals during sham exposure. No movements were detected. The authors concluded that the locus of perception of pulsed RFR is most likely within the cochlea rather than anywhere else in the head.

6.5.2 Calcium Efflux

Over the last six years, Adey and Bawin and their colleagues have reported extensively on their studies of changes of radioactive calcium ion ($^{45}Ca^{++}$) efflux from neonate chick brain preparations and isolated samples of cat cortex under very specific regimes of amplitude modulation frequencies and power densities for ELF, VHF, and UHF fields (Adey, 1975a, 1975b, 1976, 1977, 1978, 1979, 1980; Adey and Bawin, 1977, 1978; Bawin, 1974; Bawin and Adey, 1976a, 1976b, 1977; Bawin et al., 1975, 1977a, 1977b, 1978a, 1978b; Sheppard et al., 1979).

Extensive details of the experimental protocol are given in two of the papers (Bawin and Adey, 1976b, 1977). Briefly, following decapitation, forebrain hemispheres of neonate chicks were obtained by rapid dissection. The hemispheres were separated; one was used for exposure and the other as control. Each was incubated for 30 minutes in a specified physiological solution containing $^{45}Ca^{++}$. At the end of incubation, the samples were rinsed three times with nonradioactive solution. They were then transferred to new glass test tubes, bathed in 1.0 ml of solution, and exposed or sham-exposed for 20 minutes. Sets of ten brain samples (ten exposed hemispheres, ten control hemispheres) were used simultaneously. At the conclusion of exposure, aliquots of 0.2 ml of the bathing solution were taken, and radioactivity was assayed by scintillation counting. Radioactivities (counts per minute, cpm, per gram) were normalized to the mean value of counts obtained in control effluxes. All normalized data were compared (by t-test) with matched samples of control values. Adey (1977, 1978) presents data from experiments with approximately 190 chick brains for 450-MHz exposures. Power densities of 0.05, 0.1, 0.5, 1.0, 2.0, and 5.0 mW/cm^2 were used for 16 Hz amplitude modulation of the field. Statistically significant increases were seen at 0.1, 0.5, and 1.0 mW/cm^2, but not at 0.05, 2.0 or 5.0 mW/cm^2, for 450-MHz RFR amplitude-modulated at 16 Hz. In a subsequent paper, Bawin et al (1978b) described experiments aimed at a better definition of the calcium sites responding to weak electrical stimulation. Changes in efflux were studied with and without imposed electromagnetic fields (450 MHz, 16 Hz amplitude modulation, 0.375 or 2.0 mW/cm^2) to ascertain the effect of calcium concentration in the exchanging medium. Also tested were pH and bicarbonate-free solutions. They also examined modification of calcium release, by the addition of lanthanum to the bathing solution, for both no-field and with-field stimulation conditions. Efflux of $^{45}Ca^{++}$ in the standard physiological solution was the 'control" for these experiments. In any test series, each half brain was tested against the corresponding hemisphere.

The results confirmed the previous findings by Bawin and Adey that amplitude-modulated 450-MHz fields can stimulate the release of preincubated $^{45}Ca^{++}$ from isolated brain tissue. This release was significantly different, statistically, at extracellular Ca^{++} concentrations of 2.16 and 4.16 mM, but not in Ca^{++}-free solutions. The release was enhanced by addition of H^{+} (0.108 mM, as HCl), even though this did not affect the efflux in the absence of the field. Omission of HCO^{-}_{3} resulted in a decrease (not statistically significant) in efflux of $^{45}Ca^{++}$ both with and without field stimulation. Addition of La^{+++} to the HCO^{-}_{3}-free solution resulted in a statistically significant decrease in $^{45}Ca^{++}$ efflux (compared with an increase in the other cases, above) for an extracellular concentration of 2.0 mM La^{+++} for both no-field and with-field (0.375 and 2.0 mW/cm^2) situations. These results, taken together, are stated to support the hypothesis that a limited number of extracellular cationic binding sites are involved in the transaction of weak extracellular electrical events and to suggest that the electrosensitive sites in La^{+++}-treated samples are in the class of sites responsible for the field response in the "standard" solution.

Bawin et al. (1977b) have also reported on results from a preliminary study involving the monitoring of calcium efflux from the intact cortex of 12 awake, paralyzed cats. The methods were similar to those utlized in the chick brain experiments. The cats were exposed for 20 minutes to 450-MHz fields amplitude modulated at 16 Hz. Power densities were 0.375 or 1.0 mW/cm^2. Results are stated to be a clear increase in $^{45}Ca^{++}$ efflux during and following the exposure in 8 of 12 animals. However, some animals apparently responded to the presence of the experimenter during sampling. Further experiments are planned to remove the possibility of artifact and to elaborate on these preliminary findings.

Blackman et al. (1979) conducted experiments that verified and extended Bawin and Adey's findings for chick brain at 147 MHz. Treated tissue was exposed in a Crawford chamber to power densities between 0.5 and 2.0 mW/cm^2 and amplitude modulation of the carrier at selected frequencies between 3 and 30 Hz. They found a statistically significant increase in calcium efflux ($\underline{p} < 0.05$) when the frequency of modulation was 16 Hz and power density was 0.75 mW/cm^2. (Preliminary findings indicating the existence of the power window, in addition to the frequency window discovered by Adey and Bawin were reported by Blackman et al. in 1977.)

The above work on calcium efflux represents one of the very few cases in which RFR effects have been found at average power densities in a possibly nonthermal range (0.1-1.0 mW/cm^2).

The incident RFR on the rectenna is continuous-wave, but a biological entity that moves through the spatial variations of the rectenna field will experience a time-varying or modulated field. However, the likelihood that the modulation frequencies and the durations of exposure to such frequencies will give rise to the calcium efflux phenomenon is negligible.

6.5.3 Blood-Brain Barrier Effects

The existence of a "blood-brain barrier" (BBB) in most regions of the brain has been established experimentally, although its specific morphology is still conjectural. This barrier normally provides high resistance to movements of large molecules (e.g., proteins or polypeptides) from the blood vessels into the surrounding brain tissue, presumably to protect the brain from invasion by various blood-borne pathogens and toxic substances. Several investigators have reported that low levels of RFR can increase the permeability of the BBB to certain substances of large molecular weight. However, others were unable to confirm such effects, thereby rendering the subject controversial, as summarized below.

Rodzilsky and Olszewsky (1957) found that permeability changes in cerebral blood vessels could be induced by various non-RFR means, including those that produce heat necrosis. Sutton and co-workers (Sutton et al., 1973; Sutton and Carroll, 1979), who were interested in the use of RFR for selective hyperthermic treatment of brain tumors, determined the maximum temperatures and exposure durations that would not alter the integrity of the BBB in the rat. They used 2.45 GHz to induce hyperthermia in horseradish peroxidase (HRP), a high-molecular-weight protein, as a tracer that is detectable both morphologically and quantitatively. Heads of rats were heated with RFR to a brain temperature of 40, 42, or 45°C. They found that BBB integrity was diminished, in orthonormic animals (37°C), by heating the brain to 45°C for 10 minutes, to 42°C for 15 minutes, and to 40°C for 60 minutes. The corresponding durations in rats precooled to 30°C were 15, 30, and 180 minutes, respectively.

Frey et al. (1975) exposed groups of anesthetized rats to pulsed or CW RFR at 1.2 GHz for 30 minutes. For the pulsed RFR, the pulse and average power densities were 2.1 and 0.2 mW/cm^2, respectively, and the power density of the CW RFR was 2.4 mW/cm^2. Sham-exposed rats were used as controls. After exposure or sham exposure, sodium fluorescein was injected into the femoral vein. Five minutes after injection, the blood of the rat was withdrawn and the brain was removed, embedded in gelatin, refrigerated, and sectioned. The sections were viewed under ultraviolet light for fluorescence, the intensity of which was scored by the viewer. Greater fluorescence was reported for pulsed than for CW RFR, and some control specimens also exhibited slight fluorescence. The investigators regard these results as evidence that exposure to RFR alters the BBB.

Spackman et al. (1978) performed a similar investigation in mice, using fluorescein and several nonphysiological amino acids as test substances. Groups of mice were exposed to sham, CW, or pulsed RFR at 918 MHz for 30 minutes. Average power densities of 2.5 and 33 mW/cm^2 were used in both the CW and pulsed modes. Also, some mice were exposed to CW RFR at 132 mW/cm^2. After exposure, the concentration of each test substance in the brain relative to the concentration of that substance in the blood plasma (the "specific concentration") was determined. A spectrofluorometer and an automatic amino acid analyzer were used to measure the concentrations of fluorescein and the test amino acids, respectively. The specific concentrations of all substances tested in the RFR-exposed animals were found to be in the same ranges as for the controls. Subsequently, Spackman et al. (1979) used whole-body heating or i.p. injection of glycerol, urea, metaraminol, or dimethyl sulfoxide (DMSO) as alternative agents to RFR exposure. They found that heating mice to 50°C in an incubator for 22 to 25 minutes caused no apparent increase in BBB permeability to the test substances. The same results were obtained for all of the injected agents except for DMSO, which produced a significant increase in BBB permeability relative to controls.

Albert et al. (1977) also used HRP as a tracer and reported regions of leakage in the microvasculature of the brains of Chinese hamsters exposed to 2.45-GHz CW RFR at 10 mW/cm^2 for 2 to 8 hours. In control animals, extravascular reaction product was found only in brain regions normally lacking a BBB. In a later paper, Albert (1979) indicated that for hamsters and rats exposed to 2.8-GHz CW RFR at 10 mW/cm^2 for two hours, partial restoration of BBB impermeability occurs within one hour after cessation of exposure and restoration is complete after two hours. Albert believes that these changes may be clinically subacute and probably cause no lasting ill effects. It should be noted that these leakages of the microvasculature of the brain occur irregularly. During the formal discussion period following presentation of a paper by Preston at the 1978 International Symposium on Biological Effects of Electromagnetic Fields in Ottawa, 27 June 1978, Albert indicated that such leakage is observed in approximately 50% of his exposed animals and in about 20% of his control animals.

Oscar and Hawkins (1977) reported changes in BBB permeability to D-mannitol due exposure of rats to 1.3-GHz pulsed or CW RFR for 20 minutes at various average power densities. Permeability changes were measured by the Oldendorf technique; that is, 0.2 ml of a mixture of ^{14}C-labeled mannitol and tritiated water was injected rapidly into each rat's carotid artery after exposure, the animal was sacrificed 15 seconds later, and brain sections were dissected out and prepared for assays of radioactivity using a liquid scintillation counter. The ratio of counts of ^{14}C-labeled D-mannitol to counts of freely diffusible tritiated water in samples of brain tissue was normalized to a similar ratio for the injected solution. This normalized ratio, expressed as a percentage, is defined as the brain uptake index (BUI). Oscar and Hawkins found statistically significant changes in the BUI at average power densities less than 3 mW/cm^2. They also found that pulsed RFR could be either more or less effective in altering BBB permeability than CW RFR of the same average power density, depending on the specific pulse characteristics used. For pulses of long duration and high pulse power density but only a few pulses per second, mannitol permeation could be induced at average power densities as low as 0.03 mW/cm^2. Their results also indicated the possible existence of a power density "window," i.e., permeability is not altered for power densities above or below the window.

Merritt (1977) endeavored to repeat the experiments of Oscar and Hawkins, but did not obtain similar results. Data from three of his experiments indicated that hyperthermia of the brain is necessary to alter permeability.

Preston et al. (1978), using methods similar to those of Oscar and Hawkins, attempted to determine whether exposure to 2.45-GHz CW RFR increased BBB permeability to ^{14}C-labeled D-mannitol. They exposed rats to 0.1, 0.5, 1, or 10 mW/cm^2, with sham-exposed rats for controls, and found no evidence to indicate that RFR exposure increased the permeability of the BBB for mannitol.

Chang et al. (1978) used a technique involving ^{131}I-labeled albumin to investigate alterations of the BBB in dogs. The heads of dogs were exposed to various average power densities between 2 and 200 mW/cm^2. In general, no statistically significant differences were found between exposed and sham-exposed animals, but the numbers of animals used in this study were too small to ascribe a high level of statistical confidence.

Several investigators have indicated that exposure to RFR may alter the sizes of vascular and extravascular volumes and cerebral blood flow rate, thereby yielding changes in the BUI that are not necessarily related to BBB permeability alterations. Blasberg (1979) reviewed many of the methods previously used for investigating BBB changes and the problems associated with these methods. Rapoport et al. (1979) have developed a method for measuring cerebrovascular permeability to ^{14}C-labeled sucrose that yields results that are independent of cerebral blood flow rate. Oscar et al. (1979) have confirmed experimentally that local cerebral blood flow is increased in the rat brain by exposure to pulsed RFR at either 1 or 15 mW/cm^2 average power density.

Based on recent findings such as those mentioned above, little quantitative confidence can be placed in the results of early experiments on RFR-induced BBB alterations. Qualitatively, it is clear that hyperthermic levels of RFR can alter the permeability of the BBB. However, additional research using current methodology is necessary to ascertain whether chronic exposure to low levels of RFR affects the BBB. The present status of this topic was reviewed by Justesen (1980) and by Albert (1979).

6.5.4 Histopathology of the Central Nervous System

Tolgskaya and Gordon (1973) reported a number of effects of [illegible] (frequencies 500 kHz to 100 GHz) on a large number (approximately [illegible]) of animals, predominantly rats. Their so-called decimeter band (500 MHz to 1 GHz, exact frequency or frequencies not specified) is almost [illegible] below--the SPS frequency of 2.45 GHz. Pathological effects claimed for high-intensity (20 to 240 mW/cm^2) radiation included multiple perivascular hemorrhages in the brain and other organs, degeneration of apical dendrites in the cortex, cloudy swelling of cytoplasm, cytoplasmic shrinkage, formation of vacuoles, unevenness of staining, disappearance of cytoplasmic structures, fatty degeneration, decrease in ribonucleo-protein, and occasional karyocytolysis. The intensities of exposure were capable of causing death of the animals (clinical signs of hyper-thermia, temperature rises up to 42-45°C) in several minutes to several hours. Photographs of the exposure arrangement show multiple animal exposures at the same time in a room appearing not to have radiation-absorbing material on the walls. It is likely that the specific absorption rates (SARs) for individual animals under these conditions varied widely and that all effects were clearly thermal in nature.

So-called "low-intensity" exposures were also carried out. The authors define threshold field intensities for nonthermal effects ("intensity not raising body temperature") for decimeter microwaves as 40 mW/cm^2 (Tolgskaya and Gordon, 1973, Table 3, p. 56). Exposures at so-called low intensity for decimeter waves were generally at or slightly below 10 mW/cm^2 for 60 minutes daily for 10 months. Investigation of the animals by ordinary morphological methods revealed practically no vascular disorders in the nervous system. "Delicate elective neurohistological methods" (unspecified) showed disappearance of spines from cortical dendrites, the appearance of beading and irregular thickening of dendrites, swelling of cytoplasm of individual cells (with appearance of vacuoles) in the basal ganglia and hypothalamus, and focal and diffuse proliferation of microglial cells, with microglial processes showing initial signs of degeneration.

Many of these low-intensity effects are similar to those described for the high-intensity exposures. In view of the exposure levels (approximately 10 mW/cm^2), the previously described exposure arrangement, and the knowledge of the possibility of localized regions of high SAR, it seems likely that the described effects (more subtle than those of frank hyperthermia) were also thermal in origin.

Albert and De Santis (1975) have also reported changes in the hypothalamus and subthalamus of Chinese hamsters exposed to 2.45 GHz radiation either at 50 mW/cm^2 for durations from 30 minutes to 24 hours or at 25 mW/cm^2 for 14 hours/day for 22 days. Changes were not evident in the hippocampus, cerebellum, thalamus, or spinal cord ventral horn. In the discussion printed with this paper, Guy pointed out that his laboratory had measured mean SARs as high as 4 mW/g per incident 1 mW/cm^2 in animals of similar size. Peak SARs could have reached 40 to 200 mW/g in selected brain regions of Albert's animals; this range far exceeds what is normally used for diathermy treatment in 20-minute exposures of patients. Rectal temperature measurement would not necessarily reflect such high SARs in localized areas.

Albert and De Santis (1976) studied CNS histological effects in 60 Chinese hamsters exposed to 1.7 GHz radiation at power densities of 10 and 25 mW/cm^2. Cytopathology was observed after 30 to 120 minutes of exposure in hypothalamic and subthalamic areas, but not in other areas. These observed effects were also likely thermal in origin for the same reasons as above.

6.5.5 EEG Studies

Many studies have been conducted on effects on the EEG and/or evoked responses (ERs) of animals exposed to RFR. Some of these have been carried out with metal electrodes either implanted in the brain or attached to the scalp during exposure. Johnson and Guy (1972) pointed out that such metallic electrodes grossly perturb the fields and produce greatly enhanced absorption of energy (i.e., field enhancement) in the vicinity of the electrodes. Such enhancement produces major artifacts in the biological preparation under investigation. These artifacts are not to be confused with the recording artifact that is produced by pickup of fields by the electrodes and leads during the recording of EEGs or ERs while the animal is being exposed. In addition to these cautions concerning methodology, it should be noted that many EEG studies are performed on heavily sedated animals, phenobarbital being the usual drug. Hence, the responses reported do not necessarily reflect those that would be expected in normal, alert animals.

Tyazhelov et al. (1977) discussed these problems and pointed out that even for the coaxial electrode developed by Frey et al. (1968), diffraction of EM waves is still a major source of error because of the electrode's metallic nature and large dimensions. They solved the problems by developing electrodes of high linear resistance (greater than 100 kilohms/m) and by proper filtering of the recorded signal. This paper indicates an awareness in the USSR that questions may be raised about the validity of data and conclusions from many experiments involving animals with indwelling electrodes, both in the USSR and the United States.

Bruce-Wolfe and Justesen (1979) investigated the effects of microwave-induced hyperthermia on the visually evoked electrocortical response (VER) in five female guinea pigs. The VERs were recorded post-exposure from animals that had been exposed to modulated 2450-MHz RFR in a multimode, mode-stirred cavity for durations ranging from 4 to 15 minutes. Such exposures raised rectal temperature to as high as 43°C and brain temperature to as high as 41°C. The mean latency [illegible] stimulation to the P_1 peak diminished from 42.8 to [illegible] ms for cortical temperature increase of 37.0 to 40.5°C. For cortical temperatures above 41.5°C, the VER became highly variable, and above [illegible].0°C the animals died. The authors recognized the possibility of local brain damage resulting from the use of implanted metal electrodes and stainless-steel screws, and state that this aspect is being examined in further studies in the rat and guinea pig.

Dumanskij and Shandala (1974) reported changes in the bioelectric [illegible] in the brain cortex of rabbits after 60 days' exposure to RFR ([illegible] MHz, 2.45 GHz, 10 GHz). Changes (vaguely specified as "an increase in the rhythm of slow waves and a decrease in the rhythm of intermediate and fast waves") were described at 10 and 1.9 μW/cm², but not at 0.01 μW/cm². Although the rather sketchy nature of this description precludes definitive critique of these results, it appears that the use of metallic electrodes may have contributed artifacts, as described above.

In a more recent presentation, Shandala et al. (1976) reported on observations of rabbits with implanted EEG electrodes, and again claimed quite variable but statistically significant EEG changes at 10 μW/cm² exposures (2.375 GHz) for 7 hours/day for 1 month. The same questions about implanted electrodes possibly causing artifactual effects must be raised.

Goldstein and Sisko (1974) studied the EEGs of sedated rabbits to determine whether RFR exposure would evoke arousal. They used 9.3 GHz RFR at 0.7 to 2.8 mW/cm². The EEG of each rabbit was recorded for about one hour. After the first ten minutes, the rabbit was exposed or sham-exposed to the RFR for five minutes. The EEGs showed no arousal during RFR exposure but indicated alternations of arousal and sedation characteristics starting 3 to 12 minutes after exposure. However, control animals also exhibited alternations, with shorter arousal durations, rendering interpretation of these results difficult. These investigators were aware of the potential problem of metals in the pathway of the RFR and claimed to have mitigated it by using thin (0.01 inch) insulated, implanted stainless-steel electrodes. It is unlikely that this reduced the artifacts significantly, if at all. They also stated that "under everyday conditions, the EEG patterns of rabbits are quite variable. The animals oscillate between sedation and arousal unpredictably." This variability is another potential source of error in any experiments on the EEG of rabbits.

Chou et al. (1978) used implanted carbon electrodes to avoid the artifactual problems associated with metal ones. Two groups of rabbits (six animals/group: three males, three females) were exposed to 2.45 GHz, 1.5 mW/cm^2 radiation for 2 hours daily for 3 months. One group received CW, the other pulsed radiation (10 μs, 100 pps, 1,500 mW/cm^2 pulse power density). A similar group of six animals was sham-exposed. No significant differences were observed between groups at the end of three months with regard to EEG and evoked potentials.

Kaplan et al. (1978) reported that from the beginning of the second trimester of pregnancy, 33 squirrel monkeys were exposed for 3 hours/day in special cavity/cage modules to 2.45 GHz pulsed radiation at whole-body mean SARs equivalent to those resulting from plane-wave exposure to 0.1, 1.0, and 10.0 mW/cm^2 and compared with a group of eight pregnant sham-exposed monkeys. Eighteen of the exposed mothers were exposed with their offspring for an additional 6 months after parturition, and then their offspring were exposed alone for another six months after weaning. No statistically significant differences were found between exposed and nonexposed adults nor between exposed and nonexposed offspring on resting EEG and photically driven EEG parameters. (No chronically attached or indwelling electrodes were used.)

Rosenstein (1976) exposed one group of eight female rats to 10 mW/cm^2 at 425 MHz for 4 hours/day from the 12th day after breeding until parturition, and another group of 12 dams to 5 mW/cm^2 at 2.45 GHz for 4 hours/day from the 6th day after breeding until parturition. The offspring were then exposed for 92 days. Control groups having the same population numbers were used for each frequency.

Evaluation of the EEGs and the visual ERs of the offspring at 140 days of age indicated no significant difference between the exposed and control groups. (Again, indwelling electrodes were not used.)

In summary, the use of indwelling metallic electrodes in studies on the effects of RFR on the EEG and /or evoked potentials may be questioned as a procedure likely to introduce artifactual effects in the preparation under study, as well as in the recordings themselves. These artifacts may be minimized by use of electrodes appropriately designed from high-resistivity materials. Experiments in which such specially constructed electrodes were used, or in which electrodes were applied after exposure, show no evidence of statistically significant differences in EEGs or evoked responses between control and RFR-exposed animals.

6.5.6 Conclusions

In relation to the problem of exposure to microwaves within the rectenna area of SPS, the microwave hearing phenomenon is an effect only of pulsed radiation. Since the SPS radiation is continuous, the microwave hearing phenomenon has no bearing on exposure of workers. The calcium efflux phenomenon is an effect only of low-frequency amplitude modulated radiation. While humans moving under the rectenna could be exposed to modulated RFR fields, the likelihood that the rate and duration of their motion would be sufficient to produce the calcium efflux phenomenon appears negligible. Changes in blood-brain barrier have been reported at power-density levels as low as 3 mW/cm^2, but the evidence is in dispute. Histological changes in the brain have been reported at power-density levels as low as 3 mW/cm^2, but the evidence is in dispute. Histological changes in the brain have been reported at power-density levels as low as 10 mW/cm^2, but the effects may have resulted from an unusually high SAR within the skull. Microwaves have no effect on evoked potentials or EEGs. For the ordinary exposure within the rectenna area (0.01 to 1.0 mW/cm^2) there is little or no probability of an effect on either blood-brain barrier or brain histology. For the maximum exposure witnin the rectenna area (23 mW/cm^2), there could be changed in the blood-brain barrier or brain histology, but the pathological significance of these changes is not yet understood.

6.6 EFFECTS ON BEHAVIOR

The very large number and variety of behavioral studies in animals exposed to microwaves makes it difficult to present a detailed review. The papers reviewed in this section are selected as being representative of the types of behavioral studies that have been conducted. The types include microwave perception studies, effects of microwaves on learning and on performance of trained tasks, and interactive effects of microwaves and drugs on behavior.

With regard to perception of microwaves, the microwave hearing effect discussed in the previous section is, by definition, perception of microwaves. Other studies of modulated microwaves have been conducted to determine whether they can serve as a behavioral clue. King et al. (1971) showed that 2.45 GHz microwaves, modulated at 60 and 12 Hz, could serve as a cue to warn rats of impending electrical shock. The effect had a threshold of between 1.2 and 2.4 mW/g. Since the study was conducted in a cavity system, plane-wave power densities were not available; however, a reasonable estimate is that the power-density threshold would be between 2.5 and 6 mW/cm^2. Frey and Feld (1975) showed that rats exposed to 1.2 GHz pulsed at 100 to 1000 pulses per second would tend to avoid the radiation by moving into an RF-shielded area. The authors interpreted this behavior to indicate that the microwave radiation produced a noxious stimulus, but the evidence indicates that the noxious stimulus was probably the microwave hearing effect. The avoidance behavior was produced at 0.6 and 0.2 mW/cm^2 for the 100 and 1000 pulse/sec exposures, respectively, but exposure to 2.4 mW/cm^2 CW radiation did not produce any avoidance behavior.

A number of other microwave perception studies were designed to determine whether animals would avoid CW radiation as a noxious stimulus. Monahan and Ho (1977) observed that when mice were exposed for various periods to 2.45-GHz microwaves at various power densities and ambient temperatures, the mice were able to reduce the percentage of microwave energy absorbed. There was an apparent threshold of power level at which the effect would occur at ambient temperatures of 20 and 24°C, but not at 30 and 35°C. The authors attributed the effect to the ability of the animals to orient themselves in the radiation field so as to minimize energy absorption. In a subsequent study, Gage et al. (1979a) observed rats and mice by closed-circuit TV during exposure to 2.45-GHz microwaves and failed to observe orientation effects. However, comparison of the power levels and ambient temperatures in the two experiments suggests that the power density and ambient temperatures employed by Gage et al. may have been too low to produce the orientation behavior.

A second approach to the question of radiation avoidance (Monhan and Henton, 1979) involved exposing mice to 2.45 GHz microwaves at an average SAR of 45 mW/g (90-100 mW/cm^2), coupled with a sonic cue. The mice learned to turn off the microwaves by interrupting a beam of light; this was interpreted as escape behavior.

A third type of approach involved pairing microwave irradiaton with consumption of sucrose solution and subsequently testing for avoidance of the sucrose. In an earlier test (Monahan and Henton, 1977) there was no evidence that the sucrose was associated with a noxious experience. In a subsequent test (Sessions, 1979), saccharin (instead of sucrose) was paired with microwave exposure of rats. The animals developed aversion to the saccharin (indicating association with a noxious experience) at a power density of 41 mW/cm^2 or greater, but not at lower levels.

The results cited above are interpreted by the authors as indicating that microwaves are a noxious or unpleasant stimulus to the animal. However, the orientation behavior observed by Monahan and Ho (1977) appears to be, at most, thermoregulatory behavior of the animals, and the saccharin aversion appears to require relatively high power densities of the microwaves. The escape by interrupting the beam of light (Monahan and Henton, 1979) also involved relatively high power densities, and in addition, appeared to require the coupled sonic cue. Grove et al. (1979) observed that rats exposed to nearly lethal levels of microwave radiation in the absence of other cues (e.g. sonic cues) made no attempt to escape· even though the means of escape were readily available. Overall, it can be concluded that pulsed or otherwise modulated microwave radiation can be perceived readily by animals at moderate-to-low power densities, but that CW microwaves are, at best, an extremely feeble perceptual cue.

Many studies have been conducted on the effects of microwave radiation on the performance of trained tasks. Animals studied were rats, rhesus monkeys, and squirrel monkeys. Acute exposures at power-density levels ranging from 10 to greater than 100 mW/cm^2 (Sanza, 1977; de Lorge, 1979; D'Andrea, 1977; Lin, 1977; Scholl and Allen, 1979; de Lorge and Szell, 1979; McAfee et al., 1979) resulted in somewhat inconsistent results. The overall conclusion is that microwave irradiation will suppress performance of learned tasks, but that the effect depends upon power density, duration of exposure, animal species, and the demand characteristics of the behavior. The studies of de Lorge and Szell (1979) indicate that suppression of learned behavior tasks by acute microwave irradiation depends on the amount and distribution of energy absorbed by the animal.

Chronic microwave irradiation has also been reported to disrupt learned behavior in animals. Lobanova (1974) reported a weakening of conditioned reflexes in rabbits and rats, as shown by increased latency or absence of response and failure to recognize the conditioned stimulus. Power-density levels in her studies were 1 to 10 mW/cm^2. Mitchell et al. (1977) reported that rats showed an increase in locomotor activity and a disturbance of differential responding to operant behavior over a 22-week exposure at 2.3 mW/g ($\sim$ 5-6 mW/cm^2). Lebovitz and Seaman (1979), however, found no disturbance in lever-pressing performance in rats chronically exposed at up to 2.6 mW/g.

Studies of the effect of microwaves on learning are more recent and fewer. Schrot et al. (1979) investigated the effects of microwave irradiation on the ability of rats to learn a novel sequence of responses to obtain food reinforcement. Decrements of learning occurred at power densities of 5 and 10 mW/cm^2, but not at 1 mW/cm^2 or less. Gage et al. (1979b) exposed rats daily for four hours to 50 mW/cm^2 from day 6 of gestation until the age of 126 days, and found no effect of the irradiation on the learning of two tasks during the last two weeks of irradiation.

Several studies of interaction of microwave irradiation and drugs that affect the CNS have been conducted (Monahan and Henton, 1979; Thomas and Maitland, 1979; Thomas et al., 1979; Maitland, 1979). In the first study, chlordiazepoxide was found to interfere with radiation avoidance responses, but chlorpromazine and d-amphetamine gave variable results. In the other studies, the effect of a drug on animal behavior was tested, and then the effect of the drug and microwave irradiation together was tested. Microwave radiation at a power density of 1 mW/cm^2 was found to enhance the effects of dextroamphetamine, chlordiazepoxide, and pentobarbital. An interesting aspect of these studies, taken together, is that the drugs have pharmacologicaly different and opposite properties. Dextroamphetamine is a CNS stimulant, while chlordiazepoxide and pentobarbital are CNS depressants, but the microwave radiation enhances the effect in either case.

Some of the behavioral studies seem to have originated from studies in the USSR claiming that RFR had direct effects on the CNS at low power densities. The association is discussed by King et al. (1971). Evidence to support this claim from neurophysiological studies in the USA is meager, and the behavioral evidence also does not generally support the claim. The studies on microwave irradiation as a noxious stimulus do not show that the animals can perceive microwave radiation as such. The radiation-avoidance studies appear to be part of the thermoregulatory behavior of animals, and under circumstances where the environment is cold, animals will use microwave radiation as a source of warmth (Stern et al., 1979). In addition, Adair and Adams (1980) showed that microwave irradiation enhanced dermal vasodilatation in the squirrel monkey (a thermoregulatory response). The effect appeared to be mediated by the CNS, but a minimum of 8 mW/cm^2 was required to elicit the response. Disruption of performance or learning appears to have rather high power-density tresholds. Interaction of microwaves with drugs affecting the CNS appears to be the most sensitive behavioral response to microwaves, but even these studies do not prove a direct effect of microwaves on the CNS. Overall, the behavioral studies do not indicate a special effect of microwaves on the nervous system, and the mechanism of most of the results remains unknown.

In reference to the SPS, workers in the rectenna area exposed at the ordinary power density level (0.01 to 1.0 mW/cm^2) would probably not experience any effect on performance of trained tasks or speed of learning, but workers exposed at the maximum power density level (23 mW/cm^2) might experience some performance or learning decrement, particularly if the exposure is of long duration. Workers who are on medication might be subjected to enhanced action of the drugs at either the ordinary or the maximum power density level. However, there probably will not be any subjective feelings of malaise in non-medicated workers exposed at either the ordinary or the maximum power density levels.

6.7 ENDOCRINOLOGICAL EFFECTS

Microwave irradiation of animals has produced somewhat inconsistent effects on the endocrine system of mammals. In general, the effects produced appear to be related to either the heat load associated with the irradiation or the stress induced in the animals by the irradiation and, possibly, other experimental circumstances. Some effects also appear to be related to alteration of the circadian rhythm by microwaves. There do not appear to be any effects arising from direct stimulation of the endocrine system or the associated parts of the CNS.

The heat load induced by the microwave irradiation primarily affects the thyroid gland and, to some extent, the adrenal glands. Magin et al. (1977a, 1977b) observed that direct exposure of the thyroids of dogs to 2.45 GHz irradiation caused an increase in the rate of release of thyroxine. The amount of increase was dependent on the power-density level, and the authors attributed the effect to the rise in temperature of the gland. Lu et al. (1977) reported that whole-body exposure of rats to 2.45 GHz irradiation at 20 mW/cm^2 for 4 or 8 hours resulted in a decrease in serum thyroxine levels. The decrease was associated with a significant rise in rectal temperature. Exposure of rats at lower power densities or for shorter durations caused only minor changes in body temperature and insignificant depressions of serum thyroxine levels. Travers and Vetter (1978) also reported lowered serum thyroxine levels in rats exposed 8 hours/day to 2.45 GHz irradiation for 7 to 21 days. These authors claimed effects at 4 to 8 mW/cm^2, but the report was available only in abstract form and hence the data could not be reviewed.

Stress responses in rats exposed to 2.45 GHz microwaves were reported by Michaelson et al. (1975), Lotz (1976), Lotz and Michaelson (1977) and Lotz et al. (1977). These studies showed evidence of time/power-density thresholds. Rats exposed at 50 mW/g for 30 or 60 minutes showed a rise in plasma corticosterone level and a decrease in plasma pituitary growth hormone level, both of which are indicators of a general stress response. Irradiation at lower power densities for 30 or 60 minutes did not produce any similar response, but when the duration of irradiation was extended to 120 minutes, plasma corticosterone was elevated at a power density of 20 mW/cm^2 and plasma pituitary growth hormone was depressed at a power density of 13 mW/cm^2. Hypophysectomized rats and rats treated with dexamethasone did not show an increase in plasma corticosterone at any power density, indicating that the microwave effect was mediated through the anterior pituitary.

Because of the known sensitivity of the testes to heat, there has been some investigation of the effects of microwaves on gonadal function. Prausnitz and Susskind (1962) reported that mice exposed to 9.27 GHz microwaves at 100 mW/cm^2 for 4.5 minutes per day, 5 days per week, began to show testicular atrophy after 4 months of treatment. More recently, Cairnie and Harding (1979) reported that exposure of mice to 2.45 GHz irradiation at 20 to 32 mW/cm^2 for 16 hours a day for 4 days had no effect on sperm count or percent of abnormal sperm. No endocrinological measurements were reported for either study. Mikolajczyk (1974, 1975) reported that irradiation of rats to 2.86-2.88 GHz at 10 mW/cm^2 for 2 to 6 hours a day for 35 days caused a decrease in the pituitary level of luteinizing hormone. Finally, Lancranjan (1975) reported that men occupationally exposed to radiofrequency radiation at levels of up to several hundred microwatts/cm^2 showed slightly reduced sperm counts, but normal plasma levels of 17-ketosteroid and gonadotropic hormone.

Effects of low-level microwaves on circadian rhythm were reported by Czerski (1974) for the mitotic figure count of bone marrow. Lu et al. (1977) reported that the circadian rhythm of normal plasma corticosteroid levels had a confounding effect on the assessment of low-level microwave effects. A subsequent report (Lu et al., 1979) indicated that high and low power-density levels may have opposite effects on plasma corticosteroid, with the high power-density effect arising from stress and the low power-density effect arising from circadian rhythm changes.

Although most of the effects of microwaves on the endocrine system appear to be relatively straightforward and predictable from physiological considerations, there are still some details concerning the effects of microwaves on adrenocortical steroids that require further study. Part of the problem appears to arise from the uncertain nature of "stress." Heat, or temperature rise, is assumed to be a stress to the animals. However, Liburdy (1979a) observed that mice exposed to warm air to produce a temperature rise equivalent to that produced by microwave irradiation did not show a rise in plasma steroids comparable to that produced by the microwave irradiation, and did not exhibit the lymphopenia produced by the microwave irradiation. Furthermore, a number of other experiments on immunological effects of chronic microwave irradiation have produced effects at power-density levels too low to elicit stress responses in an acute irradiation situation. Finally, there is a substantial accommodation factor in eliciting stress responses. Animals that are placed in novel situations are much more prone to exhibit stress responses than are animals that have been adapted to the situation, and the use of sham-irradiated controls does not always eliminate the problem.

Workers in the rectenna area of SPS exposed at the ordinary power density level of microwaves (0.01 to 1.0 mW/cm^2) would not be expected to show any endocrine changes resulting from the exposure. If such an exposure were repeated regularly, the workers might experience some alterations in circadian rhythm, but this effect would probably not be serious. Workers exposed to the maximum power density level (23 mW/cm^2) would probably show changes in blood thyroxine levels, and possibly increases in blood adrenocorticoid levels. The probability of occurrence of such changes would increase with increasing duration of exposure.

6.8 IMMUNOLOGICAL EFFECTS

The accumulation of reports to date indicates that microwave irradiation has quite definite effects on the immunological system of mammals. Most of the reported effects were detected after exposure at relatively high power-density levels (> 10 mW/cm^2); a few have been detected following exposure to low power-density levels (∿ 0.5 mW/cm^2); and in some cases effects obtainable from high power-density exposure were not found at lower power densities. In most studies the mechanism for the effect was not investigated, and there is a fair degree of inconsistency among the separate reports.

To present a reasonable overview of immunological effects, the principal results of a sample of the studies are summarized in Table 6-3. The first four studies were investigations of the ability of microwave irradiation in vitro to act as a mitogenic stimulus for lymphocytes. This effect was first reported by Stodolnik-Baranska in 1967, was repeated (with some difficulty) by Czerski (1975) and by Baranski and Czerski (1976), but Smialowicz (1976) could not obtain a similar result. After extensive study, Baranski and Czerski concluded that the mitogenic effect depended, in a complex way, on the rise of temperature in the culture medium. On the basis of his study, one would conclude that microwaves have no intrinsic mitogenic effect on lymphocytes.

The next three studies were investigations of the effect of microwave irradiation in vivo on the number of cells of the spleen forming antibody to sheep red blood cells (SRBC). Krupp (1977) reported an increase in number of anti-SRBC cells following acute irradiation. Czerski (1975) reported the same following chronic irradiation at low power-density levels, and Smialowicz et al. (1979b) reported no effect following subchronic irradiation over a wide range of power densities. The results of Krupp (unfortunately, only obtainable as an abstract) indicated that the same effect could be produced by an increase in the temperature of the animals or by injecting them with cortisone, suggesting that the effect was a stress response mediated through the endocrine system. The results of Czerski indicated that there was an increase in the number of anti-SRBC cells after six weeks

Table 6-3

EFFECTS OF MICROWAVES ON THE IMMUNE SYSTEM

Type of Study	Author (year)	Animal Species	Microwave Frequency (GHz)	Power Density (mW/cm^2)	Duration (hr)	Periodicity	Principal Finding	Remarks
Lymphoblast Transformation In *Vitro* Irradiation	Stodolnik-Baranska (1967)	Human	3.0	7,15	-	Once	Blast transformation	Cell cultures
	Czerski (1975)	Human	-	-	-	Once	Blast transformation	Poor reproducibility
	Baranski & Czerski (1976)	Human	10.0	5-15	-	Once	Blast transformation	Temperature dependence
	Smialowicz (1976)	Mouse	2.45	10	1-4	Once	Negative results	
Sheep RBC Antibodies	Krupp (1977)	Rat	2.6	10-20	-	Once	Increase in number of antibody cells	Temp. dependence cortisone inducible
	Czerski (1975)	Mouse	2.95	0.5	2	Daily, 6-12 wk	Increase in number of antibody cells	Effect at 6 wk reversed at 12 wk
	Smialowicz et al. (1979b)	Mouse	0.425	0.63-39	1	5 days	Negative results	
Lymphocytosis or Lymphopenia	Rotkovska & Vaček (1975)	Mouse	2.45	100	0.083	Once	Lymphocytosis at 4-7 days	
	Pazderova-Vejlupkova (1979)	Rat	2.74	24.4	4	Daily, 7 wk	Lymphopenia	No effect at 1 mW/cm^2
Mitogenic Stimulation of Lymphocytes	Wiktor-Jedrzejczak et al. (1977)	Mouse	2.45	11-22	0.5-0.75	Once	Increase of F_c^+ and C^+ cells	
	Smialowicz et al. (1979a)	Rat	2.45	5	4	Daily, 35-55 d.	Increase of mitogen responding cells	Only longer exposure positive
	Shandala et al. (1977)	Rat	2.375	0.5	7	Daily, 30 d.	Reproduction in number of T cells	Full publication not available
	Huang & Mold (1979)	Mouse	2.45	5-15	0.5	Daily, 1-17 d.	Fluctuation in number of band T cells	Time-dependence responsiveness
	Liburdy (1979a)	Mouse	0.026	800	0.25	Once or 20 d.	Increase in numbers of band T cells	Same effect from corticoid injection
	Schlagel et al. (1979)	Mouse	2.45	-	0.25-0.75	Once	Increase in number of C^+ cells	Dependence on genetic factors
	Guy et al. (1976b)	Rabbit	2.45	10	23	Daily, 6 mo.	Decrease in number of B cells	
Other Studies	Liddle et al. (1979)	Mouse	9	10	2	Daily, 5 d.	Elevation of circ. antibody titer	No protection against organism challenge
	Majde & Lin (1979)	Mouse	0.148	30,#0.5	1	Daily, 3 d.	Suppression of anaphylaxis	1 hour exposure to cold gave same effect
	Liburdy (1979b)	Mouse	2.5	30,#10	0.5	Daily, 10 d.	Enhanced lymphocyte activity against graft	
	Liburdy (1979c)	Mouse	2.5	30	0.5	Once	Altered migration of T-lymphocytes	
Health and Disease	Szmigielski et al. (1975)	Rabbit	3	3	6	Daily, 6-12 wk	Reduced granulocyte response to infection	No animals died
	Pautrizel et al. (1975)	Mouse	N.S.	N.S.	6	Daily, 6 d.	Protection against lethal infection	
	Prausnitz & Susskind (1962)	Mouse	9.27	100	0.067	Daily, 59 wk	Protection against colony infection	Incidental observat. infection unintended
	Szmigielski et al. (1979)	Mouse	2.45	5,20	2	Daily, 1-4 mo.	Earlier appearance of exptl. tumors	Only 20 mW was effective

of irradiation, but that after 12 weeks of irradiation, the effect had disappeared, suggesting that the animals had accommodated physiologically to the radiation.

Rotkovska and Vacek (1975) reported lymphocytosis following a brief, intense exposure to microwaves, and Pazderova-Vejlupkova (1979) reported lymphopenia following chronic microwave exposure at 24 mW/cm^2. When Pazderova-Vejlupkova attempted to repeat the work at 1 mW/cm^2, she found no effect; the latter experiment was performed in collaboration with the USSR Academy of Sciences. Lymphopenia was also reported by Liburdy (1979a) following a single, intense exposure of mice to microwaves. The occurrence of the lymphopenia depended on a rise in core temperature of the irradiated animals; irradiation at nonthermogenic doses produced no response. However, heating the animals to the same core temperature produced only a weak lymphopenic response.

Several studies have been conducted on the number of immunologically reactive lymphocytes in the spleen or circulating blood of animals exposed to microwaves. The most extensive review of the nature and experimental manifestation of cellular immunity and of the effects of microwaves on cellular immunity is presented in the study by Wiktor-Jedrzejczak et al. (1977). The responses were somewhat variable, with the only certain conclusion being that there were effects. The results of Smialowicz et al. (1979b) and of Huang and Mold (1979) indicate that the response may depend on duration of exposure, suggesting an effect similar to that of Czerski (1975) with sheep red blood cells. The results of Schlagel et al. (1979) suggest that genetic factors may play a role in the response: mouse strains having the histocompatibility $H\text{-}2^k$ haplotype were responsive, whereas those bearing the $H\text{-}2^b$ and $H\text{-}2^d$ haplotypes were not. The results of Liburdy (1979a) indicated that the response was associated with a rise in plasma corticosteroid hormone levels and could be produced by injection of synthetic corticosteroid hormones.

Various other immunological responses have been studied. Liddle et al. (1979) observed that mice immunized against Streptococcus pneumoniae had higher circulating antibody titers against the organism after five days of microwave irradiation. This higher titer did not, however, protect the mice from challenge with a virulent strain of S. pneumoniae. Majde and Lin (1979) reported suppression of anaphylactic response following microwave irradiation. A similar effect was observed by Liburdy (1979c). Madje noted that the same effect could be produced by keeping the animals in a cold room. This observation is quite interesting in that exposure to cold is a known producer of stress. Perhaps cold exposure could be used to identify microwave effects that are stress-mediated and to distinguish between stress-mediated and temperature-mediated effects. Liburdy (1979b) observed that microwave irradiation increased the potency of lymphocytes for rejecting allografts of EL-4 lymphoma cells. He also reported that microwave irradiation selectively caused circulating T-lymphocytes to be trapped in the lungs and to be directed to the bone marrow, rather than the spleen.

Several studies have been conducted to determine whether microwave effects on the immune system alter the severity or incidence of disease. This is a relatively difficult area to investigate, and reliable, consistent results are hard to achieve. Szmigielski et al. (1975) observed rabbits experimentally infected with Staphylococcus aureus following microwave irradiation for 6 to 12 weeks. He reported that microwave-exposed animals showed a depression in peripheral granulocyte count, a depression in granulocyte reserve that could be mobilized by bacterial endotoxin, and an increased lysozyme activity of serum; he stated that the irradiated animals appeared "sicker." On the other hand, Pautrizel et al. (1975) reported that radiofrequency irradiation of mice conferred protection against an otherwise fatal challenge with Trypanosoma equiperdum. Finally, as noted above, Liddle et al. (1979) reported that microwave irradiation caused elevation of antibody titer against S. pneumoniae, but no protection against challenge by the living organism. Prausnitz and Susskind (1962) observed during long-term chronic microwave irradiation of mice that the irradiated mice appeared to have more resistance than controls did to a pneumonia infection accidentally introduced into the colony. However, this was an incidental observation, and not a planned experiment. Szmigielski et al. (1979) reported that microwave irradiation of mice at 20 mW/cm^2 and simultaneous treatment with diethylnitrosamine or 3,4-benzpyrene led to earlier appearance of tumors from these carcinogens. When the microwave irradiation was given at 5 mW/cm^2, however, it had no "promoting" effect on carcinogenesis.

In summary, microwave irradiation has effects on the immune system of mammals. Some of the reported effects were obtained at low power-density levels, but most of the studies were performed at relatively high power densities, and in some cases effects obtained at high power density were not found at lower power densities, suggesting the possibility that power-density thresholds exist. Some of the reults indicate immunosuppressive effects, some indicate immunostimulative effects, and some indicate that the state of the immune system depends on the duration of exposure or the time--in relation to irradiation--when measurements were taken. The existing evidence indicates that the immune system effects are probably mediated through the effect of the radiation on the endocrine system, involving the general adaptation syndrom to stress. It appears doubtful at present that microwaves have any direct stimulatory effect on the cells of the immune system.

From the viewpoint of the SPS, workers exposed to the radiation at ordinary power density levels (0.01 to 1.0 mW/cm^2) would probably exhibit no changes in immunological response, particularly if the exposure occurred rather irregularly. If the exposure occurred regularly on a five-day-a-week schedule, there might be some changes in immune responsiveness. Workers exposed to the maximum power density (23 mW/cm^2) might show some changes in immune responsiveness. Again, regular exposure would be more likely to produce changes than sporadic exposure. Evidence for potential hazard to humans from immunological changes brought about by microwave exposure is inconsistent, but there is presently no strong evidence that harm would probably result.

6.9 BIOCHEMICAL, PHYSIOLOGICAL, AND CELLULAR EFFECTS

A number of reports have been published indicating that microwave irradiation has specific direct effects on biochemical processes, enzyme systems, cell membranes, and muscle contraction. This section considers some of the completely published reports on these phenomena.

In a study of mice exposed to 2.45 GHz in the dose-rate range of 0 to 44 mW/g, Ho and Edwards (1977) found that mice decreased their oxygen consumption and metabolic rate during exposure. The decreased oxygen consumption appears when the radiation dose rate exceeded the basal metabolic rate, and returned to normal when the irradiation was discontinued. Phillips et al. (1975) exposed rats to 2.45 GHz microwaves at 4.5, 6.5, and 11.1 mW/g for 30 minutes. Colonic and skin temperatures were elevated at all dose rate levels, and oxygen consumption was reduced at 6.5 and 11.1 mW/g, but not at 4.5 mW/g. The reduced oxygen consumption at 11.1 mW/g persisted for more than five hours after cessation of exposure. In a more recent study, Lovely et al. (1979) found that rats exposed to 2.45 GHz radiation at 5 mW/cm^2 for 10 hours per day reduced their food and water consumption without significant reduction in body weight. Both of these effects can be reasonably attributed to metabolic adaptation to the heat load induced by the irradiation.

Deficis et al. (1979) exposed mice to 2.45-GHz microwaves continuously for 60 hours at 3-4 mW/cm^2. They reported that the mice had elevated levels of serum triglycerides and serum beta-lipoproteins two hours after termination of the radiation. There was some indication that the effect might be power density-dependent. This result may be related to the study of Pazderova et al. (1974), who reported that workers in television and radio transmitting stations had decreased serum albumin levels and increased serum alpha- and beta-globulin levels. The report noted that although the changes were significant, the values were still within the normal human range and the workers appeared to be in good health.

Although claims have been made that microwave irradiation alters the permeability of cell membranes, Liu et al. (1979) found no effect of microwaves on the membrane permeability of rabbit, human, and dog erythrocytes other than what could be attributed to a rise in temperature of the suspending medium. Similarly, Allis and Fromme (1979) found no effect of microwave irradiation on adenosine triphosphatase, an enzyme involved in maintaining the sodium-potassium balance of erythrocytes.

The calcium efflux phenomenon and the effects of microwaves on the blood-brain barrier (discussed in Section 6-5) might also be considered effects of microwaves on membrane permeability. The calcium efflux phenomenon dependsupon a low-frequency amplitude modulation of the radiofrequency signal, and hence is probably not an intrinsic property of the microwaves. The effects of microwaves on the blood-brain barrier remain in dispute at present, but the evidence indicates that the effect is subject to a power-density threshold.

Several reports have been published indicating that microwaves affect smooth muscle contraction. McArthur et al. (1977) found that 960-MHz irradiation of post-pyloric segments of rat gut at 1.5 to 5.5 mW/g caused an increase in peristaltic activity. The increased activity could be blocked by treatment of the segment with atropine. On the other hand, Whitcomb et al. (1979) found no effect of 1-GHz microwaves on contraction rate of isolated segments of rat gut.

Reed et al. (1977) found that exposure of the isolated rat heart to 0.960 GHz microwaves at 2 mW/g for 10 minutes caused a slight bradycardiac (~ 5 to 10% reduction below controls. Pretreatment of the isolated hearts with atropine and propanolol to block the autonomic nervous system also blocked the microwave-induced bradycardia. Phillips et al. (1975) observed a bradycardia in intact rats following exposure to 2.45 GHz microwaves for 30 minutes. At an SAR of 4.5 mW/g the effect was insignificant; at 6.5 mW/g, it was mild and returned to normal within two hours; at 11.1 mW/g, it was pronounced, and tachycardia developed after two hours. In addition, seven out of ten rats at 11.1 mW/g and two out of ten at 6.5 mW/g exhibited irregular heart rhythm. The rhythm returned to normal within one hour. They calculated that the plane-wave power density corresponding to 6.5 mW/g in these animals was 31 mW/cm^2.

In summary, microwave irradiation produces a metabolic adaptation to heat load and a change in serum lipids and proteins. Continuous-wave microwaves have no effect on cell membranes of membrane bound enzymes except that produced by temperature rise, but modulated microwaves at low modulation frequency may affect membrane function. Reports of effect on smooth muscle contractions appear to be inconsistent and, in any case, are reported only for isolated preparations and not for intact animals. Microwave irradiation at high power density levels can produce temporary bradycardia and irregularities in cardiac rhythm.

Workers in the rectenna area of the SPS would probably show changes in serum protein levels similar to those reported by Pazderova et al. at both the ordinary (0.01 to 1.0 mW/cm^2) and maximum (23 mW/cm^2) power-density levels of exposure. There is no evidence that this effect would have any significance for human health. In addition, workers exposed to the maximum (23 mW/cm^2) power density level would experience a significantly increased heat load which could lead to several biochemical and physiological effects that are part of the normal physiological response to heat load. In addition, depending on the SAR in various locations in the human body, workers exposed to the maximum power density (23 mW/cm^2) could experience temperatures elevated considerably above the average at specific sites, which could lead to special compensating mechanisms. Finally, workers exposed to the maximum power density (23 mW/cm^2) might occasionally experience slight, but temporary, bradycardia and irregularities of cardiac rhythm.

7 ECOLOGICAL ISSUES

The ecological effects of the solar power satellite system within the rectenna area can be considered under several headings, e.g., effects of construction activity, effects of presence of the rectenna, etc. This section will consider only the possible effects of the microwaves from the satellite on the biota within the rectenna area.

The effects of microwaves on the biota can be considered from two points of view: effects on climate in the region, and direct biological effects of the microwaves on the biota. Since most of the microwave energy will be absorbed by the rectenna and converted into electric energy, the residual incident power density available for altering the regional temperature (about 1 percent of 1 to 23 mW/cm^2) will be essentially trivial, and there would be no expected effect on climate. Likewise, the rectenna design provides for 98 percent transmission of solar energy down to the ground, so there will be no perceptible shading effect of the rectenna.

Biological effects of the microwave radiation can be considered for three regions: the region underneath the rectenna, the region between the exclusion fence and the rectenna, and the region above the rectenna where insects, birds, and other vertebrates may fly around or perch on top of the rectenna. The region directly underneath the rectenna will be exposed to power density levels of between 0.01 and 0.23 mW/cm^2, the region between the exclusion fence and the rectenna will be exposed to power density levels of between 0.1 and 1.0 mW/cm^2, and the region above the rectenna will be exposed to 23 mW/cm^2.

There is a general lack of information about the effects of microwaves on the diverse species of the animal and plant kingdom. Based on the biological studies summarized in Section 6, there are few documented effects at power density levels significantly below 1 mW/cm^2; hence it might be reasonably expected that there would be no significant effects on the biota residing and remaining below the rectenna. Between the exclusion fence and the rectenna (0.1 to 1.0 mW/cm^2) there might be some biological effects, particularly in the region immediately adjacent to the rectenna. The nature of these effects cannot be predicted, but it should be noted that, because of the penetrating nature of microwaves, there could be some effects relating to the distribution of water within the soil and the plants and insects immediately next to the rectenna.

Above the rectenna, biological effects of the microwaves would definitely be expected, but again, the exact nature of the effects carnot be predicted. Gary and Westerdahl (1978) have summarized a variety of reported effects caused by exposure of insects to RFR. The direct effects ranged from unrest to death, depending on the level and duration of exposure and the species studied. More recently, in a preliminary report submitted to EPA, they noted that brief exposures to microwaves at power densities ranging up to 50 mW/cm^2 had no significant effect on the ability of honey bees to return to their hives. Effects of mammals, birds, and other vertebrates are somewhat difficult to predict. A preliminary report on effects of microwaves on birds (Arthur D. Little, 1980) consists mainly of a description of experimental design and facility construction. Probably, one of the major hazards would be to birds in flight over the rectenna area. These birds would be already subject to a substantial heat stress from the act of flying, and the additional heat burden from the microwaves might overload thermal compensating mechanisms in these animals. The problem of vertebrates on top of the rectenna is complicated by the possibility that during cold weather these animals might seek the top of the rectenna as a region of comparative warmth. This could create a problem of reduced energy absorption and conversion within the rectenna.

In summary, the major effects of the microwave radiation on the biota of the rectenna area will most likely be found in the region above the rectenna. Presently available data are insufficient to make exact predictions of the biological and ecological effects in this region.

8 MISCONCEPTIONS

Several misconceptions regarding the bioeffects of RFR continue to be expressed in popular accounts outside the scientific literature on the subject. Those accounts tend to be sources of some confusion to the intelligent nonspecialist. The following are representative examples.

The distinction between RFR and ionizing radiation is often not made; consequently, the known hazards of the latter are linked--by implication--with exposure to RFR. In essence, ionizing radiation (which includes ultraviolet light, X-rays, and the emissions from radioactive materials) has sufficient quantum energy (see Section 5.1) to expel an electron from a molecule, leaving the latter positively charged and thereby strongly affecting its interactions with neighboring molecules. Ionization can alter the functions of biological molecules fundamentally and often irreversibly. By contrast, the quantum energies of RFR are so much smaller, that their primary* effect is to agitate molecules rather than ionize them. Also, RFR-induced agitation ceases as soon as exposure to RFR is halted. At low RFR intensities, the heat that such agitation represents is well accommodated by the normal thermoregulatory capabilities of the biological entity exposed, and therefore such effects are generally reversible. At high RFR intensities, the thermoregulatory capabilities may be inadequate to compensate for such effects, thereby leading to thermal distress or even irreversible thermal damage. In summary, a single quantum of ionizing radiation that is absorbed by a molecule alters the properties of that molecule and thereby may profoundly affect the function of the biological constituent involved, whereas the concurrent absorption of many quanta of RFR is necessary to cause biologically significant effects.

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*The possibility of long-range quantum interactions, discussed in Section 5.1.3, is not excluded; however, evidence of their occurrence in live animals is sparse as yet, and there is no evidence that such effects could be harmful if they do occur.

It follows from the discussion above that even if an effect is produced by RFR, that effect may not necessarily be deleterious to the entity involved. As an example of a nonhazardous biological effect, normal levels of light (a form of electromagnetic radiation having quantum energies above those of RFR but below those of the ionizing radiations mentioned previously) are absorbed by the skin and converted into harmless heat and, of course, such absorption in the eyes is necessary for vision. Nevertheless, it should be stated that one of the reasons why the levels of allowable exposure of humans to RFR are generally lower in Eastern European countries is their philosophically-based assumption that every effect produced by RFR is potentially harmful--a view not generally shared by Western countries.

Concerned people often ask whether quarantees can be offered that chronic exposure to low levels of an agent such as RFR will have no deleterious effects many years in the future. The frequent ingestion of small quantities of certain substances that are stored in the body can result in a large, potentially harmful total dose. Unlike such substances, RFR energy continually absorbed at low incident power densities (dose rates) is readily dissipated and does not accumulate toward the equivalent of RFR energy absorbed at high incident power densities. This is one of the basic reasons why there are threshold power densities for the various RFR bioeffects.

Accounts of purported deleterious effects of RFR exposure on humans sometimes appear that, on investigation, are found to be either specious or not due to RFR exposure. Moreover, some of these accounts persist or are repeated well after investigations have shown them to be incorrect. Examples of such occurrences include the videoterminal cases discussed in Section 6.4.2 and the often reported Moscow Embassay story discussed in Section 6.1. In the videoterminal situation, the level of RFR from the terminals was found to be unmeasurable relative to the environmental level. Regarding the Moscow embassy, residents did indeed suffer various illnesses, but the occurrence of such illnesses could not be ascribed to RFR exposure. The intelligent nonspecialist should examine such popular accounts with caution.

9 EVALUATION

The potential impact of the solar power satellite radiation can be considered from four points of view: effects on workers in the area of the rectenna system, effects on workers in space-building or maintaining the satellite, effects on the general population, and effects on the ecology within and outside of the rectenna.

A worker traversing the region between the exclusion fence and the edge of the rectenna would be exposed to between 0.1 and 1.0 mW/cm^2. Once underneath the rectenna and proceeding toward the center, he would be exposed to approximately one percent of the incident power density, with a maximum of 0.23 mW/cm^2 at the center (USDOE and NASA, 1978). Thus, the full range of ordinary exposure of the worker in the area of the rectenna system would be 0.01 to 1.0 mW/cm^2. The review of biological effects in Chapter 6 reveals only a few reported effects at power densities in this range:

- Clinical studies on occupational exposure of persons to radiofrequency radiation over a broad range of frequencies (Klimkova-Deutschova, 1974; Sadcikova, 1974; Pazderova, 1971). Pazderova reported that the workers were in good health, and that the only change observed was a statistically (but not clinically) significant rise in plasma protein levels. The results of Sadcikova and of Klimkova-Deutschova have internal inconsistencies that indicate limited application to SPS.
- The radiofrequency hearing effect. Since this effect involves only pulsed radiation, it is not relevant to SPS.
- The calcium efflux effect, reported by Adey and co-workers. This involves amplitude-modulated radiation, and hence is not applicable to SPS, which will be CW.
- The microwave-avoidance effect, reported by Frey and Felt (1975). This, again, involves pulsed radiation, and is

not applicable to SPS, for reasons stated above.

- The drug-microwave interaction effects, reported by Monahan and Henton (1979), Thomas and Maitland (1979), Thomas et al. (1979), and Maitland (1979).
- The reduced sperm counts in persons occupationally exposed to radiofrequency radiation, reported by Lancranjan (1975).
- Immunological effects reported by Czerski (1975) and by Shandala (1977a).

The medical significance of the non-pulsed effects noted above remains in doubt, but the possibility exists that some of them could appear in some of the workers under the normal conditions of exposure. The highest level of exposure would occur in traversing the space between the exclusion fence and the edge of the rectenna (0.1-1.0 mW/cm^2), so if any problems are anticipated, most of them might be avoided by transporting the workers in shielded vans.

A worker would be exposed to the full incident power of SPS (1.0 to 23 mW/cm^2) only if he had to climb on top of the rectenna or enter an open area where part of the rectenna had collapsed. A number of the biological studies have shown effects in this power-density range. Although few, if any, of the reported effects necessarily imply hazard to humans, prudent measures for the protection of workers under those circumstances of exposure may be appropriate, pending acquisition of more information on effects of RFR on humans.

For workers in space, the incident power densities are likely to be of the order of watts per cm^2. This level is much too great for a human to tolerate for any length of time. Therefore, vehicles, space suits, and other paraphernalia used by workers in the main beam area will have to be provided with adequate RF shielding, including shielding for the eyes.

For members of the general public outside the exclusion boundary, the incident power density will be 0.1 mW/cm^2 or less. There are no reliable reports that acute or chronic exposure to such power densities will have any adverse effects on humans or the flora and fauna of this region.

In evaluating the potential biological and health effects of microwave radiation from SPS, the biological effects reported in mice and rats have been treated as if exposure of humans at the same power density levels would produce similar effects. However, as discussed in Chapter 5, mice and rats have body dimensions rather close to the wave length of 2.45 GHz microwaves, and, compared to man, the SAR in mice and rats will be about 10 times greater at this frequency. Thus, in assessing effects on humans there is a conservative safety factor of 10, which will allow for uncertainties in various factors, such as power density thresholds, species differences, and others.

Ecological effects may be considered separately for species at ground level and airborne biota. For ground level species, the power density of RFR will range from 0.1 to 0.23 mW/cm^2 under the rectenna and from 0.1 to 1 mW/cm^2 between the boundary fence and the rectenna (USDOE and NASA, 1978). There is no current evidence that these power density levels will adversely affect the ecosystem in this region; indeed, over most of the region, the rate of energy deposition will average less than the diurnal temperature cycle. Airborne biota may enter the region in or above the rectenna and remain there for significant periods of time. At present there is no evidence one way or the other that such exposure would adversely affect such species.

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11 GLOSSARY

AF	audiofrequency
ANSI	American National Standards Institute
BBB	blood-brain barrier
BUI	brain uptake index
CB	citizens' band
CM	cochlear microphonics
CNS	central nervous system
cpm	counts per minute
CW	continuous wave
DMSO	dimethyl sulfoxide
DOE	U.S. Department of Energy
EEG	electroencephalogram
EKG	electrocardiogram
ELF	extremely low frequency
EMF	electromagnetic fields
EMR	electromagnetic radiation
EPA	Environmental Protection Agency
ER	evoked response
FCC	Federal Communications Commission
HEW	U.S. Department of Health, Education and Welfare
HFAL	high frequency auditory limit
HRP	horseradish peroxidase
ISM	industrial, scientific, and medical
MPTS	microwave power transmission system
NBS	National Bureau of Standards
NIEMR	nonionizing electromagnetic radiation
NIOSH	National Institute of Occupational Safety and Health
OSHA	Occupational Safety and Health Administration
prf	pulse repetition frequencies
RFR	radiofrequency radiation

SAR	specific absorption rate
SPS	satellite power system
SRBC	sheep red blood cells
UHF	ultra high frequency
VER	visually evoked electrocortical response
VHF	very high frequency
WHO	World Health Organization